

# American Journal of Orthodontics and Oral Surgery

FOUNDED IN 1915

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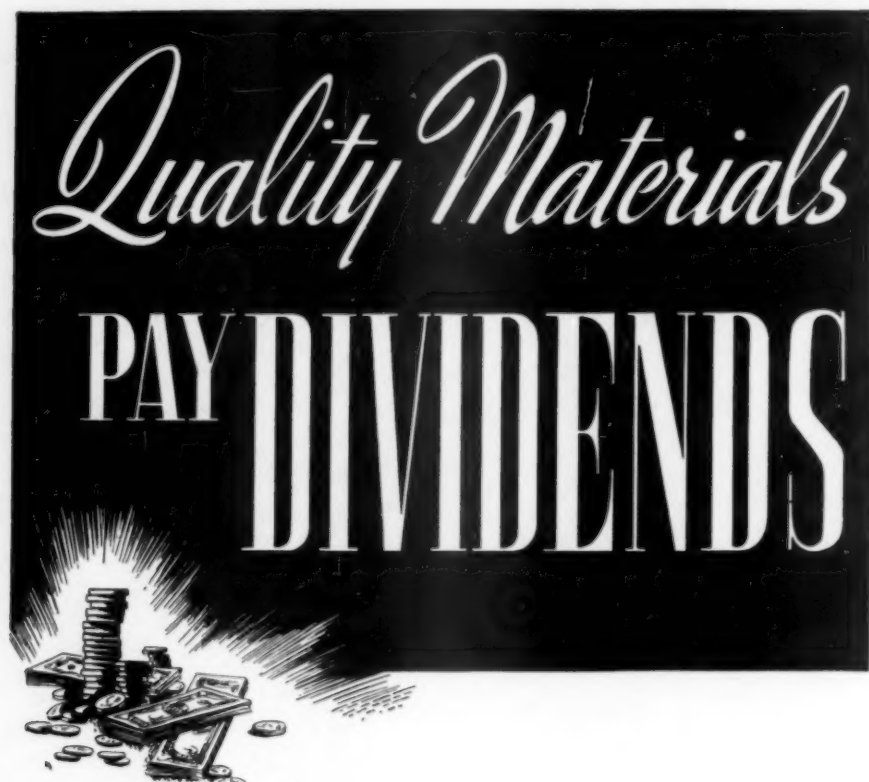
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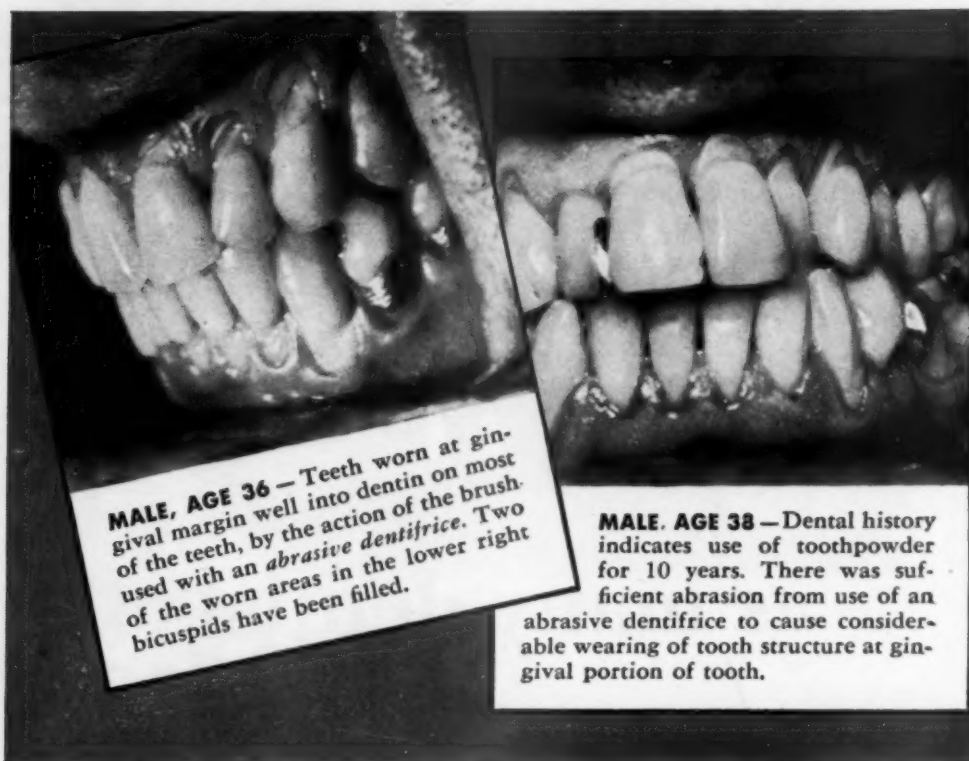
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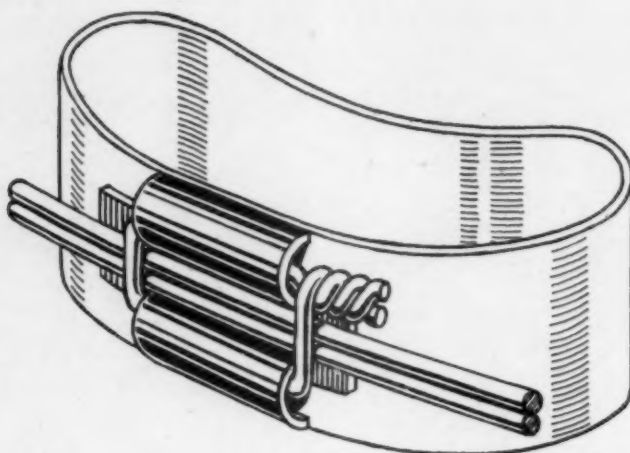
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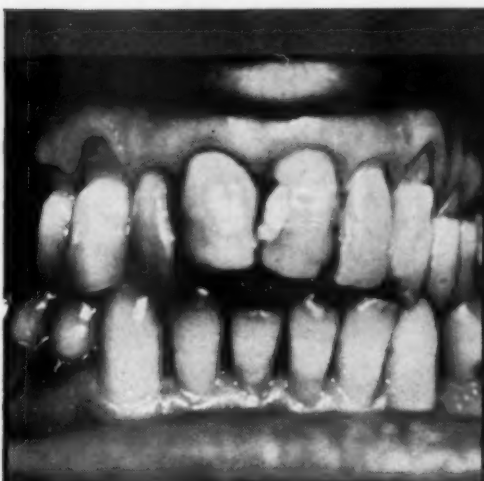
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Dental Institute School of Dentistry, University of Pennsylvania*Third Edition. Octavo, 498 pages, with 86 engravings. Cloth, \$7.00.*

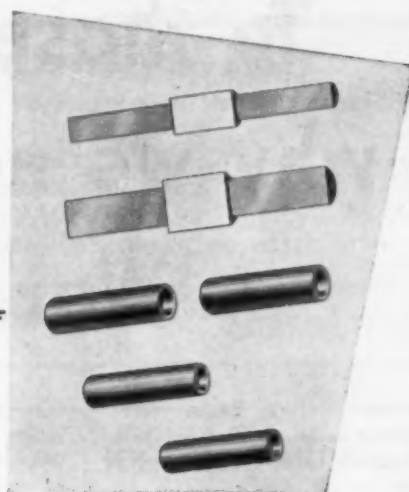
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## Original Articles

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### THE PHILOSOPHY OF THE TOOTH POSITIONING APPLIANCE

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H. D. KESLING, M.D.S., LA PORTE, IND.

---

THIS new appliance grew out of a desire to create some simple appliance that would influence all of the teeth to flow into their best possible position with relation to one another without any interference from bands or wires, that would be effective under functional forces, that would produce arch form in accordance with type, that would further attain the desired harmony between facial features and tooth arrangement, and that would serve as a retainer to conserve all the advantages gained above. As this appliance was developed, it was found to be most practical for the final artistic positioning and retention of the teeth after basic treatment had been accomplished.

In the last few years we have heard much about lack of bony base, mesial drift, bimaxillary protrusion, bimaxillary bony retrusion, and so forth. All of this has been expressed because of a realization of a lack of harmony and balance between the teeth, the jaws, and the features of the face. Perhaps no two operators would agree upon a certain pattern or profile as a typically beautiful face. It is quite encouraging that those teeth that have sufficient space in the denture to assume an upright position over their bony bases produce a pleasing profile and dentures of which the teeth are stable under functional forces. Perhaps this will be the media by which orthodontists in general will finally agree to some extent as to what constitutes balance and harmony of the dentures and facial contour.

Except in the presence of abnormal muscular habits, teeth that are unhampered by proximal contacts or inclined plane interference tend to assume positions which are stable as well as in balance with one another and with the immediate tissues surrounding them. Therefore, the diagnosis and plan of treatment of the average orthodontic case is greatly simplified when we accept our limitations of bone development and leave in each arch only those teeth

---

Read before the Fall Meeting of the New York Society of Orthodontists, Nov. 14, 1944.

which will have sufficient space to be positioned upright and properly rotated, with correct proximal contacting. Then with the mechanics we have at hand, we can place the maxillary denture in its proper relation to the mandibular. With the exception of cases with exaggerated bone deformities, orthodontic cases have a very favorable prognosis when treated in this simple and realistic manner. It would be ideal to have a case treated from start to finish with an appliance that did not interfere with the proximal contacting of the teeth nor increase their mesial-distal dimensions. To date, the profession has not



Fig. 1.—Basic treatment. Right, original model. Left, basic treatment model.

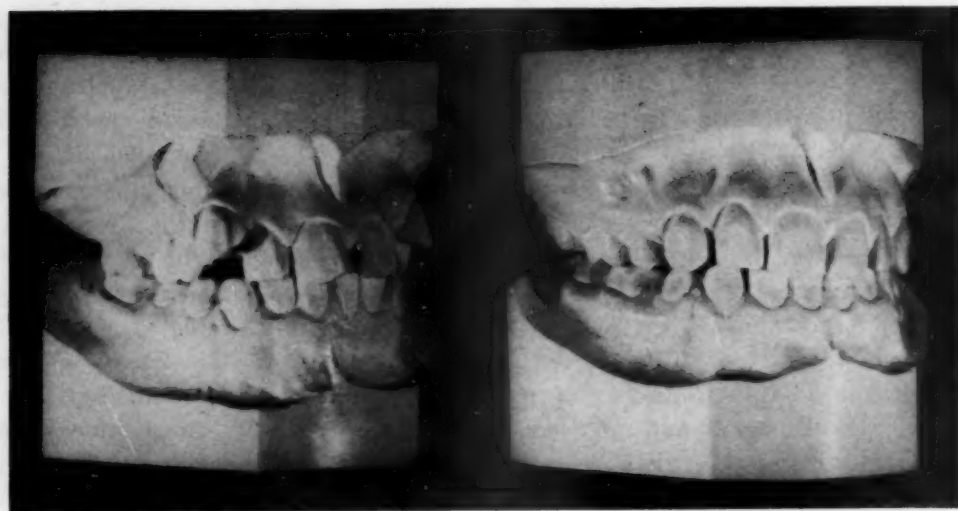


Fig. 2.—Basic treatment. Left, original model. Right, basic treatment. Note spaces.

been favored with an appliance that would adequately control the teeth that required major movements without banding or capping these teeth. We have in the "Tooth Positioning Appliance" an active treating appliance for the final artistic positioning of the teeth as well as an effective retaining device. This appliance allows the teeth to flow into their most ideal position without interference from bands, caps, or wires. Also, this appliance is most effective under functional forces.



The proved practicability of the Tooth Positioning Appliance is for the final artistic positioning and retention of the teeth of cases that have already had basic treatment completed with a conventional type appliance. Basic treatment need only be carried until each tooth is properly rotated and is approaching its desired position. Arch form need not be ideal, slight spaces may remain, overbites may still be exaggerated, and mesial-distal or buccal-lingual relationships of the maxillary teeth to the mandibular need not be perfect so long as the cusps are starting into their proper inclined plane relationships.

At this point all of the bands and wires are removed and impressions are taken immediately. From the impressions two sets of models are prepared from hard plaster. The art portions of these models should be carved and proportioned like those of any display or study models of orthodontic cases. One of the models is used to make the setup and the other is used as a control. The teeth are dissected from the setup model and are rearranged in wax on these bases to the desired arch form, axial positioning, and occlusion. This gives the operator a chance to express himself in detail as to the arch form and tooth positioning he would prefer for each patient. All orthodontic treatment is tooth positioning, but here is a practical reason for positioning with the hands plaster teeth which are an exact reproduction of the crowns of the teeth in the mouth.

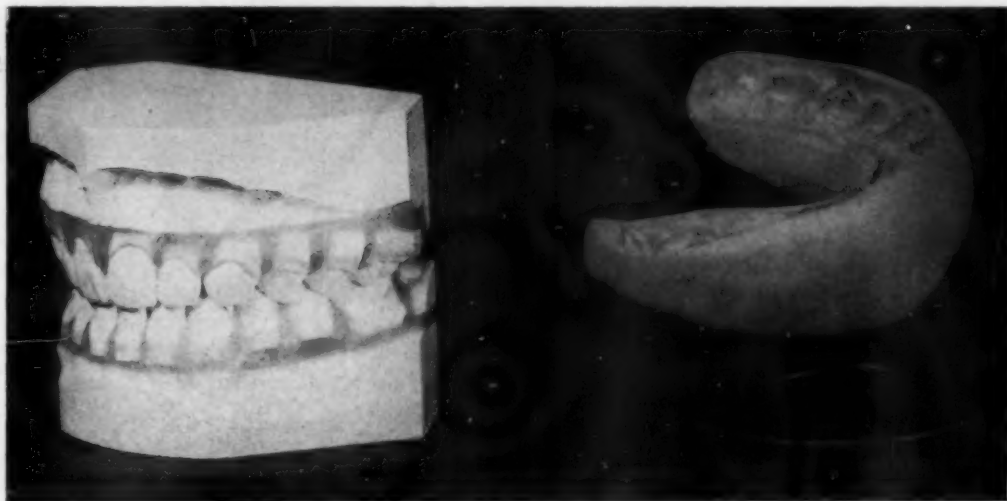


Fig. 3.—Setup and positioner. Left, setup model. Right, Tooth Positioning Appliance.

When completed, this setup model is used as a pattern over which the Tooth Positioning Appliance is constructed. The setup models are articulated on an anatomic articulator and the bite is opened to the physiologic rest position. The Positioner is a one-piece pliable rubber appliance that is made to completely fill the freeway space between the upper and lower dentures as well as to cover the labial, buccal, and lingual surfaces of both the maxillary and the mandibular teeth.

If basic treatment has been properly accomplished, each tooth will have sufficient space in the arch. The material of the Positioner allows it to stretch over the teeth, and while it is being worn its resiliency influences each tooth

toward its position in the predetermined pattern, or setup. Experience has shown that in this way arch forms may be modified, slight rotations may be accomplished, and axial position influenced.

In the past after orthodontic treatment has been completed, the average orthodontist has depended upon Nature to settle the teeth into positions of balance and harmony. Examination of a cross section of orthodontic cases throughout the country reveals that this procedure has been disappointing, even where treatment has been carefully executed.

When the Positioner is applied at the end of major tooth movements, it takes advantage of the fact that in this condition the teeth are most susceptible to its gentle forces. The teeth are unstable from previous manipulation and respond to the influence of the Positioner very readily. Not only does the Positioner maintain the advantages gained by the conventional treatment, but the teeth are actually influenced toward more harmonious and stable positions through its wearing.



Fig. 4.—Changing arch form. Left, basic treatment model. Center, setup model. Right, model after treatment with Positioner.

Orthodontic service is certainly a personal service, and we can personalize it even more by developing a distinctive arch form and tooth arrangement according to type for each individual under treatment. With the predetermined pattern it is possible for the operator to shift the teeth within reason into any desirable position, and, in proportion to his artistic ability, to give each case a distinctive, artistic touch.

Every orthodontist has hoped to develop for each of his patients an arch form according to type. In the main this has failed, because, in a high percentage of the cases treated, arch forms have been produced that would allow the teeth to assume positions which would least resist the desired tooth movements. Thus, arch forms frequently have come to be a result of expediency rather than a true development according to type. In fairness, we must give credit to the rare orthodontist who takes the trouble and succeeds in developing each arch form in accordance with type after major tooth movements have been achieved.

Not infrequently, teeth of individuals under orthodontic treatment are tipped into abnormal axial positions. This is especially true in the molar and premolar areas where the teeth are invariably tipped buccally. With the Tooth Positioning Appliance, this condition can be corrected in the setup, and in the mouth

the Positioner will influence the teeth, not only by reducing the arch width, but also by the functional forces working through the occlusal surfaces of the teeth, their roots being thrown buccally and their crowns lingually. Teeth so positioned with the Tooth Positioning Appliance will more nearly approximate the axial positioning of the same teeth in nonorthodontic normals.

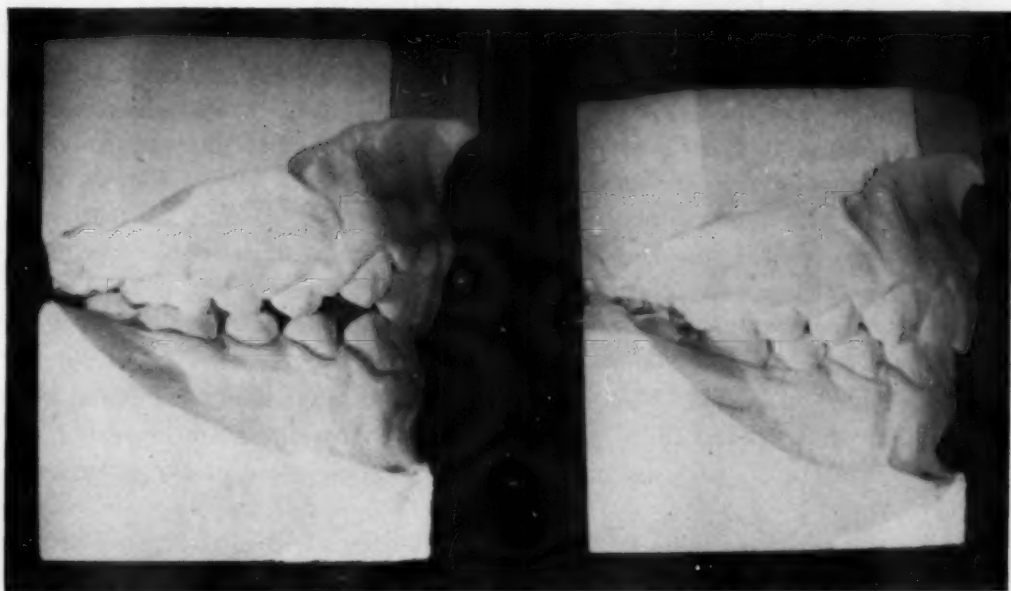


Fig. 5.—Mesial-distal correction. Left, basic treatment model. Right, model after correction with Positioner.

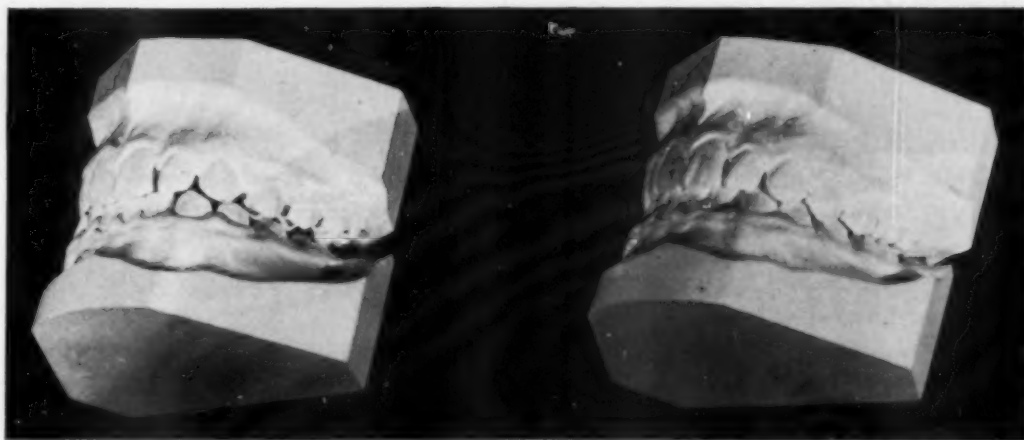


Fig. 6.—Buccal-lingual correction. Left, basic treatment model. Right, model after correction with Positioner. Note space closure.

As basic treatment is completed in severe Class II, Division 1 cases, the maxillary anteriors often assume a lingual axial inclination. This position is inevitable because of the distance the incisal edges of these teeth must travel lingually in order to reach Class I relationship to those of the mandibular incisors. When using the Tooth Positioning Appliance it is quite convenient to exaggerate the labial axial inclination of these teeth on the setup, and

through the appliance influence them into their normal positions. This same condition will often prevail in cases where it is necessary to eliminate some of the dental units in order to complete basic treatment properly.

When using the Tooth Positioning Appliance for the final positioning of the teeth, it is not necessary that all of the teeth completely interlock in inclined plane relationship before basic treatment is discontinued. If the maxillary buccal teeth are approaching their normal relationship both buccolingually and mesiodistally to their antagonists in the mandibular arch, the teeth can be forced into the Positioner, and it will influence all the teeth toward their complete interdigitation. If it is necessary to shift the mandible laterally or mesially in order to engage the Positioner, the proper force for positioning these teeth will be brought into play when the patient, after seating his teeth into the Positioner, carries the mandible back into its normal position. The functional forces will add greatly to reduce this type of discrepancy, if the patient is not lazy and works against any shift of the mandible created by the Positioner.

There are some types of major tooth movements that the conventional type of appliance has utterly failed to produce. Deep overbites are corrected not by the depression of the anterior teeth but by the elevation of the other teeth to their line of occlusion. There surely are cases of closed-bite malocclusion in which the most favorable treatment would be the depression of the anterior teeth rather than the elevation of the posterior teeth. Since the pressure required to elevate teeth is very light compared with that necessary to depress teeth generally speaking, all conventional types of appliances elevate the posterior teeth in these closed-bite cases rather than depress the anterior teeth.

When dealing with such cases the intelligent arrangement of the teeth on the setup would be an almost end-to-end relation of the anterior teeth. A Positioner processed from such an arrangement and worn by the patient would not only throw all the occlusal forces onto the anterior teeth, but also through the elasticity of the appliance would give additional "kick" to this depressing action. No other appliance previously available to the profession has such possibilities. Only as the anterior teeth are depressed would the Positioner influence the posterior teeth to any great extent. Certainly the most active force would be toward the depression of the anterior teeth.

Open-bite malocclusions have been difficult to handle with the conventional type appliance, and although in cases carefully treated the anterior teeth have been brought into the line of occlusion, this is usually entirely accomplished by the elongation of the anterior teeth. Invariably the faces of individuals with open-bite malocclusion are long and the mandible has the appearance of being depressed even though a few of the posterior teeth are in occlusion. The ideal treatment of such cases would be the depression of these posterior teeth and not the elongation of the anteriors. To date, there is not sufficient practical evidence that this can be accomplished, but certainly the Positioner offers the best possibilities of such a correction. It would be expecting too much to think that the Positioner, even under functional forces, would accomplish this result. The most favorable results so far have been accomplished with the



Positioner in place, and with a headgear being worn to which powerful elastic forces were connected with a chin cup. As in the closed-bite cases, the pressure of the occlusal forces as well as that of the elastic force from the headgear and chin cup were at first entirely directed against the teeth that should be depressed. The elasticity of the Positioner between the maxillary and mandibular posterior teeth offered an ideal additional force for the depression of these teeth.

The Tooth Positioning Appliance has tremendous possibilities as a splint for the reduction of fractures of the maxilla or mandible. This field is almost entirely unexplored. In one case, however, where there were multiple fractures of both the maxilla and mandible, the Tooth Positioning Appliance has been used successfully for their reduction. In this case the Positioner was worn in conjunction with a rigid headgear connected by elastic force to a chin cup.

The Positioner has many uses other than final positioning and retention. Major tooth movements could be accomplished with a series of Positioners by changing the teeth on the setup slightly as treatment progresses. At present this type of treatment does not seem to be practical. It still remains a possibility, however, and the technique for its practical application might be developed in the future.

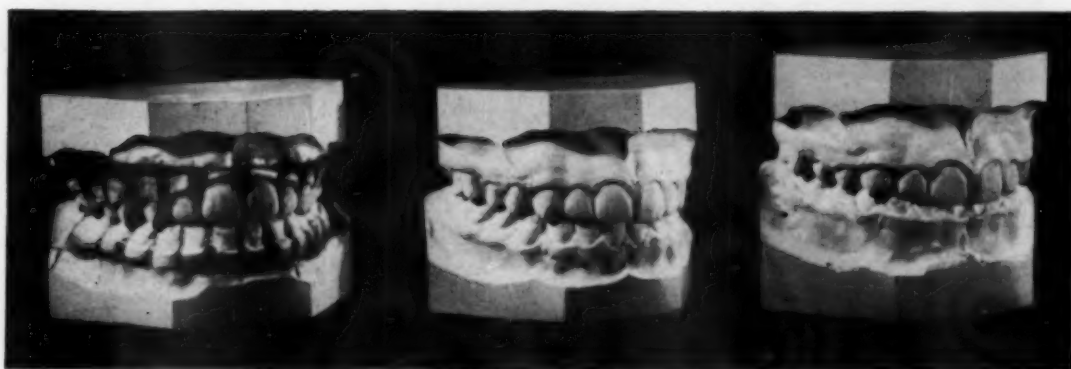


Fig. 7.—Reduction of closed-bite. Right, basic treatment model. Left, setup model. Center, model after treatment with Positioner. Note reduction of closed-bite.

In selected cases it has been practical to use one or more Positioners to direct the eruption of the permanent teeth as well as to make slight corrections of teeth already erupted without the use of any conventional appliance. There have been several closed-bite cases that have been carried to a successful conclusion without the use of any appliance other than the Tooth Positioning Appliance.

The Positioner is an ideal retaining appliance, because it not only retains the arch form and tooth positioning within the arch, but also retains the correct relationship between the maxillary and mandibular arches. It will go into place and function even though there has been a slight relapse of the case. When in place it has the proper stimulus to iron out the relapse and again position the teeth as they were arranged in the predetermined pattern. As the conventional types of retainers are rigid, they do not allow for slight changes which are inevitable in any denture. The Positioner is flexible and accommodates itself to these changes that accompany the settling of the teeth.

The Tooth Positioning Appliance has been used, and has possibilities for extensive use in the future, as an appliance to stabilize teeth of individuals who have had orthodontic treatment. It can be of equal benefit for cases that have not had treatment but which are prone to drift into traumatic malocclusion perhaps through lack of function. By using this new technique of final positioning of the teeth, it is possible to remove the conventional type of appliance from four to six months earlier than is practical under the usual form of treatment. Besides reducing the operator's chair time, the patients appreciate the shortening of treatment. When the Positioner is properly worn, each tooth is being forced toward its best possible position, not only in relation to the teeth of its own arch, but also in relation to the teeth of the opposite arch. Slight spaces are closed, moderate rotations are adjusted, maxillary and mandibular discrepancies are corrected, and proper interdigitation of the maxillary and mandibular teeth is achieved. Axial positioning is changed, not only by the pressures exerted on the buccal, lingual, and labial surfaces of the teeth, but also by the functional forces exerting pressure on the occlusal surfaces of the teeth. This is especially true of the posterior teeth.

The day of prolonged wearing of orthodontic appliances is past. The major tooth movements necessary to properly accomplish the basic treatment of most orthodontic cases can be completed in about twelve months, if the active treatment is undertaken at the most opportune time. Many cases can have the bands on and off in from six to eight months, providing that the final positioning is to be accomplished, not by bands and wires, but with the Tooth Positioning Appliance.

910 INDIANA AVENUE.

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## RESTORATION OF FUNCTION THROUGH EARLY CORRECTION OF MALOCCLUSION

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**D**URING any meeting such as the present one (devoted to the discussion of orthodontics), frequent reference is made to growth and development. Growth is generally taken to mean an increase in bulk by proliferation. Development, as defined by Conklin, is the progressive and coordinated differentiation of the organism under the influence of heredity and environment, and interplay of these factors is responsible for changes in relative proportion.

The important thing to remember in the interpretation of the following material is the division of both growth and development into prefunctional and functional stages. During prenatal life heredity is the controlling factor in

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Read before the New York Society of Orthodontists, Nov. 13, 1944, New York, N. Y.

determining the pattern of growth, but function assumes an increasingly important role in subsequent growth and development. It is logical to assume, therefore, that any disturbance of function may have an adverse effect on the growth and development of jaws and should be corrected as early as possible.

With these thoughts in mind, I shall present condensed reports of four cases in which growth and development have been inhibited at a very early age due to disturbances in function. It is customary in a case report to give the history, etiology, diagnosis, prognosis, and treatment. The history in these cases is necessarily very short since they were seen so early, in one instance before all the deciduous teeth had erupted. And although we may speculate on the etiology, it is impossible to determine an etiological factor on the basis

Fig. 1.

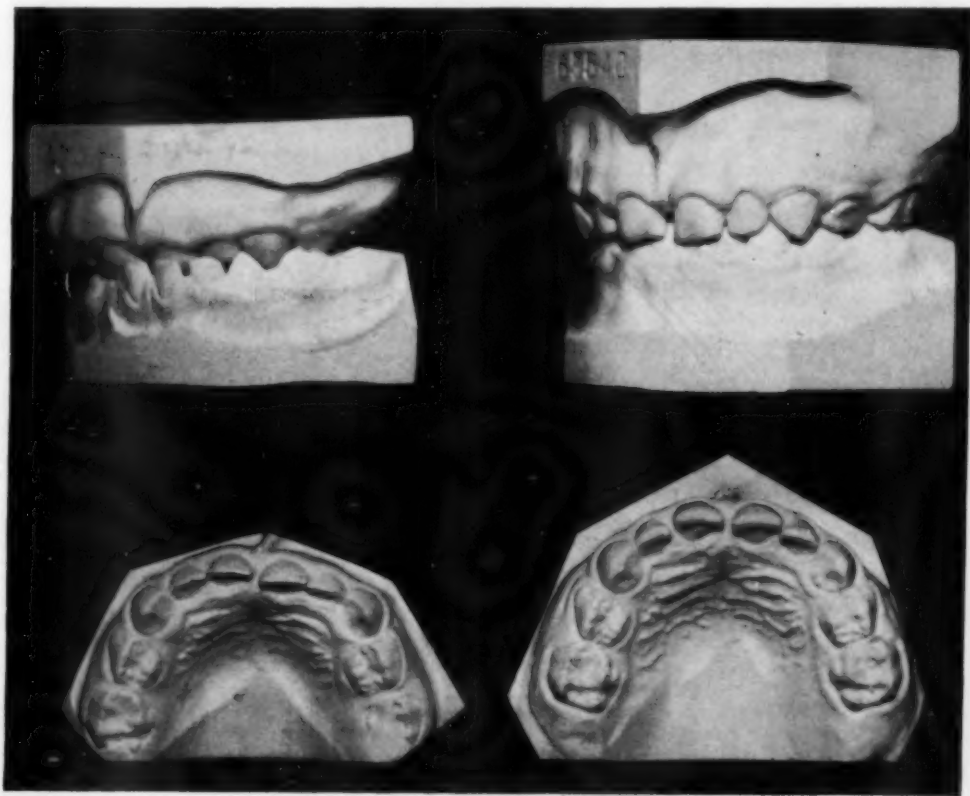


Fig. 2.

of actual cause and effect. It seems necessary, then, to classify them as true anomalies of either hereditary or congenital origin. The prognosis is good if we define as our objective an occlusion which is normal for the age of the patient, bearing in mind that final results depend upon such factors as continued normal function, nutrition, and general health. Treatment consists of two parts: the mechanical, using the simplest type of appliance of very light construction, and the myofunctional, both during treatment and later through transitional stages.

The first case (Fig. 1) is of a little girl, 2 years and 9 months of age, in apparent good health, whose history seems negative as far as any causative factor is concerned. There seems to be some original deviation from the normal in the form of the maxilla, which caused the incisors to assume this relation. Classified as Angle Class I with retruding incisors, it is a simple procedure with a plain labial wire of light gauge to correct the arch form and allow the incisors to assume a relation in which they may function normally. Active treatment was postponed until the fourth year owing to delayed eruption of the second deciduous molars, and correction was completed in three months. (Fig. 2.)



Fig. 3.



A.

Fig. 4

B.

This allows the jaws to develop for three or four years under the influence of a normal function before the deciduous incisors are lost. To be sure, a normal deciduous denture does not insure a normal permanent one, but the chances are better than for one which is in abnormal function for several years of active growth.

The next case (Fig. 3), one of the opposite extreme, is of a boy 4 years of age. This patient had been under observation for several months with the condition becoming more



Fig. 5.

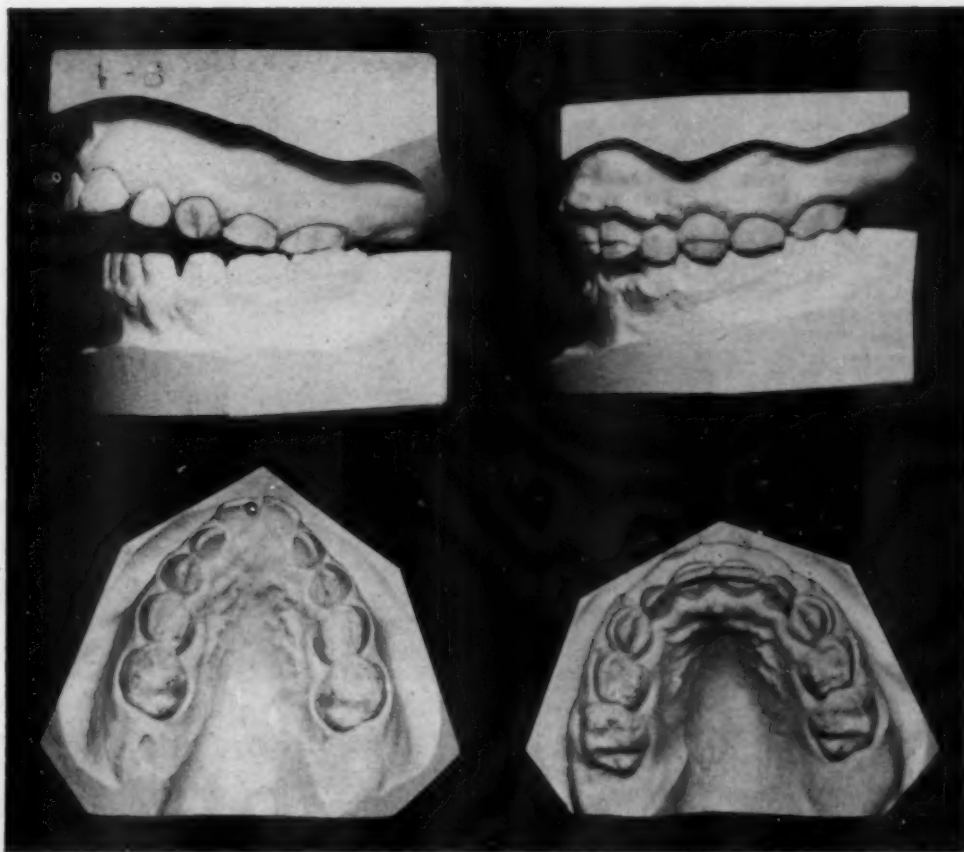
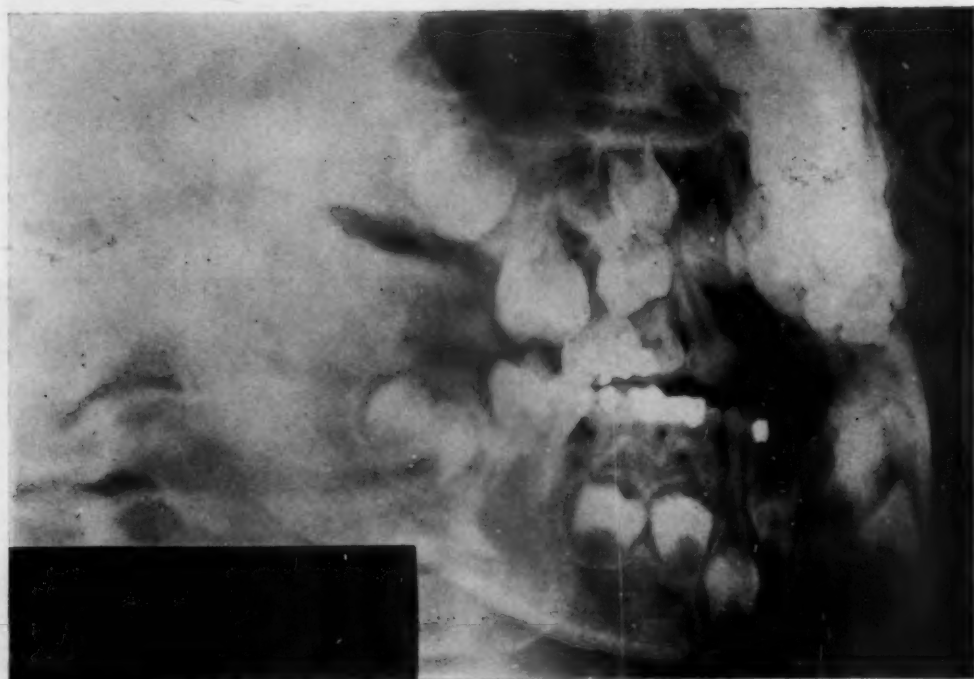


Fig. 6.



Fig. 7.

A.



B.

Fig. 8.

pronounced, until it was decided to start treatment. It appears that the habit of lip-sucking had aggravated an already serious Class II condition, causing further recession of the mandible and flattening of the lower anteriors as well as changes in the soft tissue of the lips. (Fig. 4, *A* and *B*.) The case responded quickly to simple therapy, and normal function was restored in a short time. It is important to follow up cases of this type for some time, however, using myofunctional therapy to overcome the distortion of the soft tissue due to lip-sucking, and to retrain the muscles to conform to the corrected occlusion. The progressive nature of these cases makes it more important that correction be made early, in order that the jaws may develop under the influence of normal function during the years of rapid growth.

The third case (Fig. 5), is of a girl of 4 years, small for her age but in good physical condition. The arches are extremely narrow, the maxillary arch being contracted in the molar region so that the maxillary molars occluded in palatal relation to the mandibular molars. The classification was distocclusion with open-bite. Her parents reported a finger-sucking habit, but the frequency and the force exerted did not seem to indicate that this was the principal causative factor. The patient also breathed through the mouth and this may have been the effect rather than the cause of the contraction. A few months of treatment with a junior appliance was sufficient to close the bite and develop the arches so that normal function was possible. One important reason for treating this type of case as early as possible is the tendency to develop secondary habits of lip and tongue, which aggravate the condition and are most difficult to break when they become confirmed at an early age. (Fig. 6.)

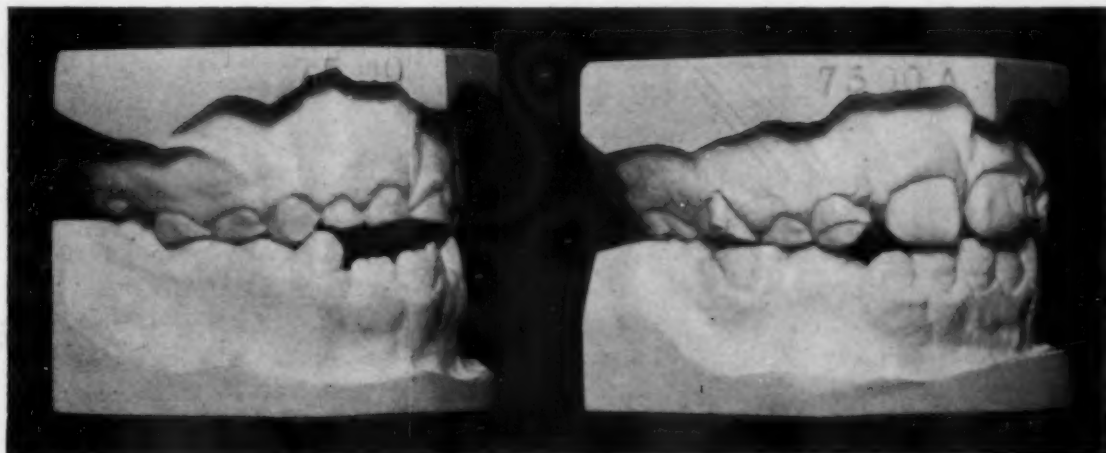


Fig. 9.

In the fourth case it appears that the growth has not kept pace with development. The patient is a boy slightly overweight and subject to almost continued colds. The permanent teeth are quite large (incisors), and there seems to be considerable discrepancy between tooth material and bone structure. This lack of space had caused the six-year molars to become impacted behind the second deciduous molars in a manner that made it impossible for them to erupt. (Fig. 7.) The importance of the six-year molars in the developing arch made it necessary to treat this condition and bring the molars into a functional occlusion. This was accomplished by means of a lingual wire and finger springs. Further treatment will be necessary as other teeth develop; but the objective of a functional occlusion has been accomplished at this stage. Incidentally it has been stated that basal bone is not affected by function. (Fig. 8.) This may be true in older patients, but in younger children, such as seen in the x-ray, there seems to be little of the bone which is not actively growing and, therefore, under the influence of function. (Fig. 9.)

## SUMMARY

It is not necessary or desirable to treat every case of malocclusion that is presented. Many, if not most, of these children of 3 to 6 years can be safely put under observation in order that the tendency of growth can be noted. A fair percentage will be seen to improve as Nature seeks to correct some earlier period of retarded growth. Others may require the correction of a habit which is obstructing the normal course of development. Still others, such as the cases shown here, require slight mechanical corrections before normal function can be established and the forces of normal occlusion can exert their influence. Practically all of these cases suffer from low and improperly balanced muscle tone, and myofunctional therapy is used both during treatment and afterward to overcome this deficiency. As the span of attention is short in these younger cases, appointments must be kept brief and appliances simple; however, if these conditions are observed, the preschool children will be found to be among our most cooperative patients.

60 CHARLESGATE WEST

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CORRECTION OF MALUNION OF A MANDIBULAR FRACTURE WITH  
DISFIGURING MALOCCLUSION

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RECOVERY from a mandibular fracture is generally uneventful. There are, however, instances in which the broken fragments knit in malunion. In such a situation there is an unfavorable realignment in the structural relationship of the bony fragments. As a consequence, the conformation of the mended mandible differs substantially from its "prefracture" contour. Its relationship to the temporomandibular articulation, to the maxilla, and to the face also is changed. Obviously, a functional disturbance and a facial deformity of varying severity must occur. Thus, it is inevitable that there would be disappointment with the outcome of the treatment for the fractured mandible.

## CASE REPORT

R. R., a woman, aged 28 years, was referred to us. She sought relief from the mutilation of the lower part of her face, especially the chin disfigurement (Fig. 1, A). She sought correction of her altered occlusion (Fig. 1, B) with its masticatory malfunction and the restricted mandibular movements. She complained of the difficulty she experienced in enunciating distinctly. Her general health apparently was good. Six months previously, in an automobile accident, she sustained "mandibular fractures, both on the left side—one in the ramus, the other in the body near the mental foramen; and as far as could be ascertained a horizontal fracture in the maxilla at the base of the nasal spine."

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A.



C.



B.



D.

Fig. 1.—A, Front view malunion present. Lower third of face disfigured; right side sloping medially, left side bulging; chin deviated toward left.

B, Teeth in centric occlusion showing altered occlusion. Indicators show median line.

C, Front view after correction of malunion. Note favorable esthetics.

D, View of teeth in centric occlusion. Note the corrected occlusion and space for a full-sized tooth. Indicators show median line.

An oral examination disclosed a badly mutilated occlusion. Her left mandibular premolars and molars occluded buccally to their maxillary antagonists. The left mandibular first incisor bit labially to the maxillary left second incisor, while, continuing to the right, the remaining mandibular teeth retrogressively occluded lingually to their maxillary opponents so that the lingual surfaces of the right maxillary premolars and molars barely occluded with the buccal surfaces of the corresponding mandibular teeth. In the maxilla there were missing the left second premolar, left first and third molars, and the right first and third molars. In the mandible all teeth were present except the left canine, and the left second incisor which was extracted during the healing period immediately following the fracture. The space between her left first incisor and first premolar measured 3 mm. (Fig. 2, A). Both these teeth were mobile, with the gingivae receded and their root surfaces exposed. The excursive movements of the mandible were restricted.

The lower part of her face was disfigured, the right side sloped medially while the left side bulged. Her chin was displaced toward the left (Fig. 1, A). Considering the deformity, her discouraged attitude was understandable.

Our roentgenograms showed union with overriding mandibular fractured ends (Fig. 3, A). Because of the malunion there was a definite diminution in the size of the mandible and a modification in the shape of the mandibular arc.

The contention often heard that union of the fractured parts was of primary concern and occlusion secondary did not seem compatible with the sequelae in this case. In view of the prominence of the face, the physical casualty is most important, yet it seemed subordinated to the hazard to the psyche. It was also apparent that by remedying the physical deformity a favorable reaction in the mental state of a normal, healthy individual would be practically spontaneous.

A study of the situation included the medical history, photographs, roentgenograms, mouldage, casts, etc., from which it was learned that prior to the accident she had a good functional occlusion, complicated only by missing teeth. At that time there was no facial deformity. As a sequel to the fracture treatment there was a severe malocclusion and an interference with mastication. The inclination and direction of the mandibular teeth indicated that the malrelation was basically in the supporting interstitial bone. It was obvious that the union of the fractured segments took place with these parts in rotated positions.

Orthodontic methods alone could not correct the malocclusion and facial deformity. Neither could surgical methods alone correct the malocclusion and facial deformity. The problem of oral and facial rehabilitation required a cooperative procedure in which the surgeon and the orthodontist would utilize and coordinate their respective resources. Thus, the *modus operandi* called upon each specialty to contribute toward the preoperative preparation, operative procedure, and the postoperative care.

By surgically fracturing the mandible for the correction of the deformity there arose the problem of reduction, fixation, and stabilization of fragments. It was apparent that the shape, number, position, and condition of the maxillary and mandibular teeth were unfavorable for direct attachment for splints. It was also observed that the displacement of the anticipated fragments may best be restored to normal by gradual reduction. There was also the possibility of the need for a bone graft.

With these thoughts in mind, we planned the use of the maxillary and mandibular splints to be constructed and inserted in anticipation of the surgery. Except for the insufficient number and shape of the teeth present, the construction of the maxillary appliance presented no complication. In order to improve our anchorage and to facilitate retention, orthodontic bands with spurs were constructed to be cemented on key maxillary teeth—the left and right canines, and the left and right second molars. For the maxillary splint a Winter Fracture Splint (a ready-made appliance with intermaxillary spurs equally spaced) was contoured and adapted to the cast of the maxillary teeth, impressions of which had been taken for our preliminary studies.

The designing and construction of a mandibular splint was of much concern since the malocclusion and disfigurement of the face were due to the malunion of the mandibular fragments and especially since the mandibular teeth were also unfavorable for direct attachment of a splint. Our ultimate aim was to restore these fragments to their "prefracture" location.

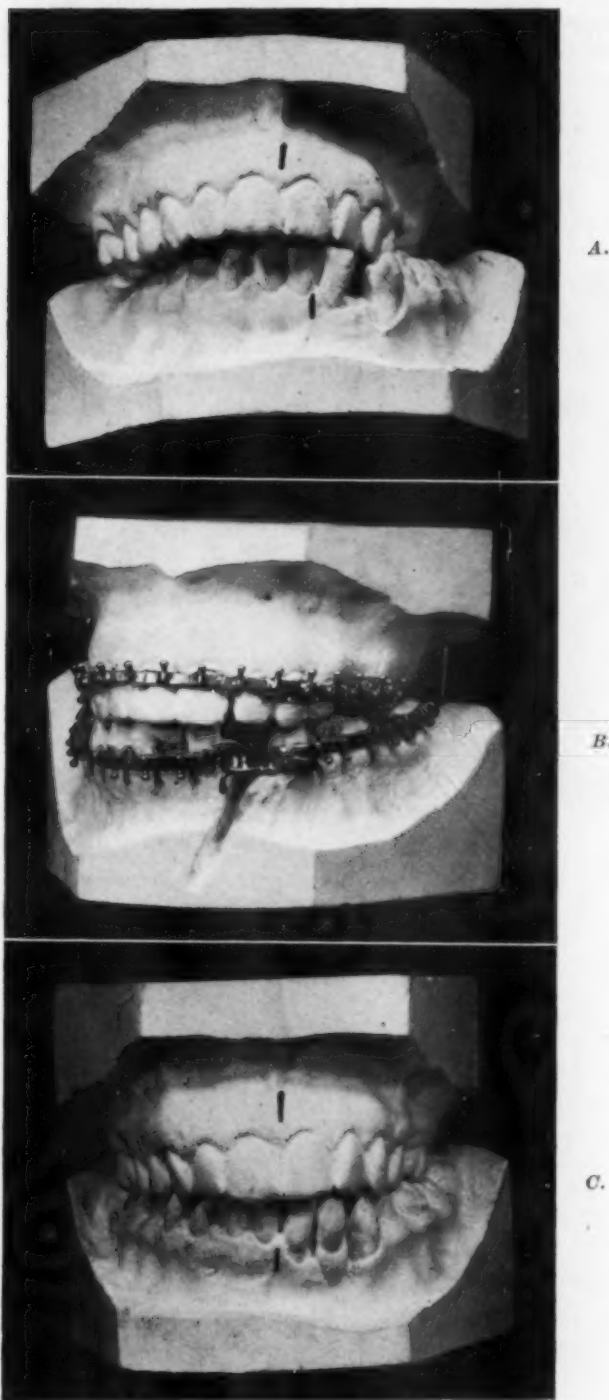
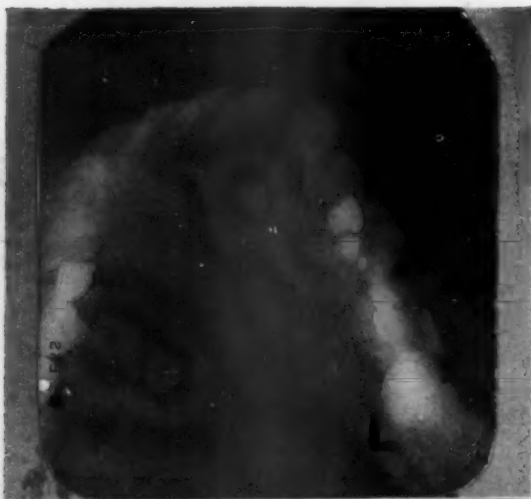


Fig. 2.—A, Casts of maxillary and mandibular teeth before refracturing of mandible. Left mandibular segment in buccoversion; right segment in complete linguoversion. Indicators show median line.

B, Articulated casts of maxillary teeth with reset mandibular segments. The mandibular cast represents corrected functional occlusion. Splints adapted to casts. Mandibular splint is shown in three sections. Midsection is removable.

C, Casts of maxillary and mandibular teeth after corrective treatment with artificial left mandibular second incisor in position. Indicators show median line.

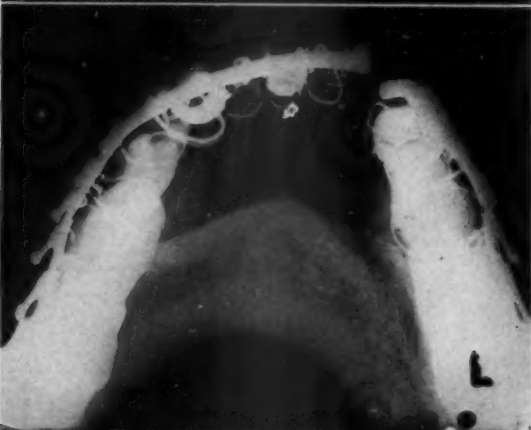
A.



B.



C.



D.

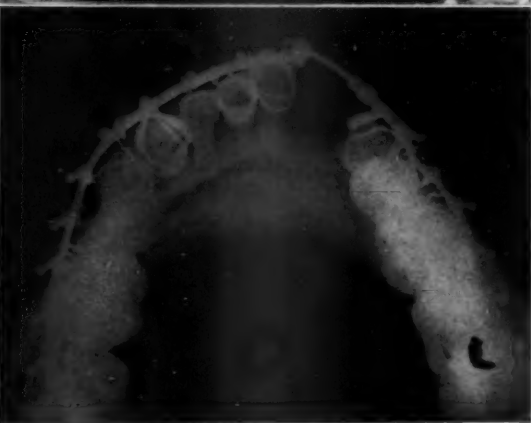


Fig. 3. (See opposite page for legend.)



The design of this splint in conjunction with the maxillary splints, therefore, had to provide firm fixation for the splint and to stabilize the surgically fractured fragments throughout the callus-forming period. It also had to be easily adaptable should conditions appear at the time of the osteotomy which called for a modification of the splint in length or direction without affecting or thwarting our objective. Therefore, orthodontic bands with spurs also were used for the mandibular teeth to improve the unfavorable anchorage, together with a Winter Fracture Splint which we modified to suit our special needs. To recover the occlusion that we believed existed prior to the accident, the cast of the mandibular teeth was broken in the region of the original fracture. These parts were reset and articulated to the

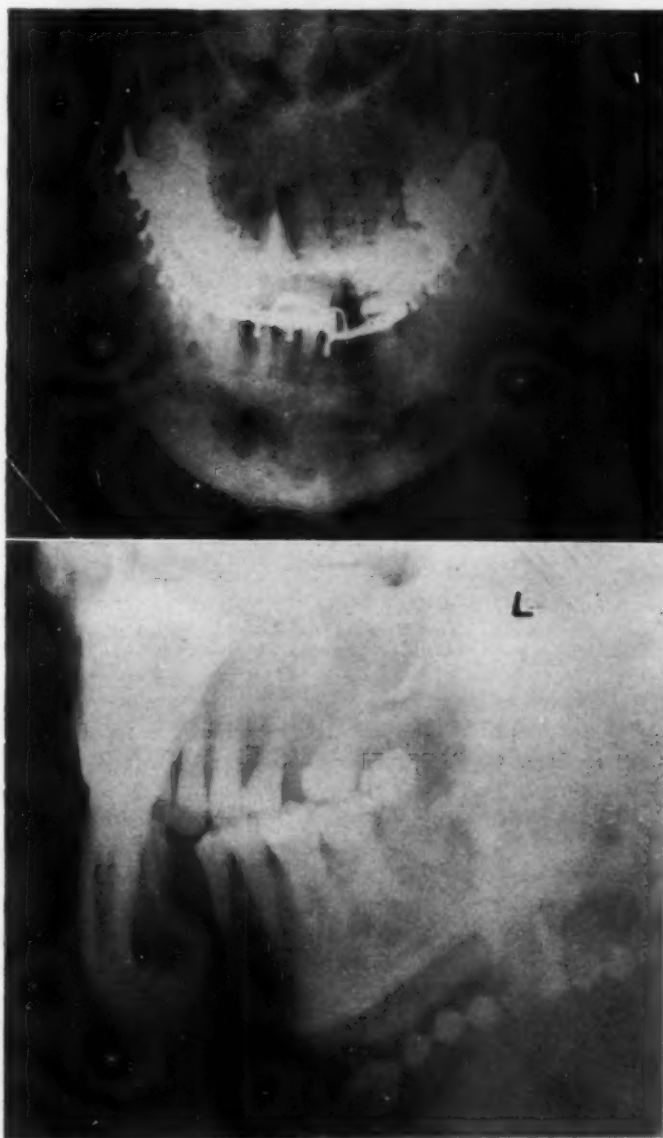


Fig. 3.—*A*, Occlusal roentgenograms showing overriding ends of fracture segment.  
*B*, Occlusal maxillary roentgenogram with splint in position.  
*C*, Occlusal mandibular roentgenogram with left and right end splints in place before operation.  
*D*, Occlusal mandibular roentgenogram showing the complete three-sectional splint in place after the osteotomy.  
*E*, Posteroanterior roentgenogram showing maxillary and mandibular splints in place. Note the midsection completing the splint assemblage.  
*F*, Left lateral roentgenogram showing alignment of segments of refractured mandible with increased space for artificial substitute after fracture splints were removed.

maxillary cast so that a good functional occlusion was approximated (Fig. 2, *B*). In this new alignment a larger space was provided between the left first incisor and the left first premolar. A fracture splint adapted to this cast obviously could not be inserted in the mouth prior to the operation and be expected to fit on the mandibular teeth (Fig. 3, *C* and *D*). To overcome this difficulty a three-sectional appliance had to be constructed, two sections of which would be inserted in anticipation of the operation and the third, a removable section, would be inserted during the operation. Thus, the desired alignment would be gotten by completing the splint assemblage with a minimum of manipulation after the osteotomy.



Fig. 4.—View of corrected occlusion of teeth after osteotomy, with splint on teeth and intermaxillary rubbers in place.

To construct the lower splint a Winter Fracture Splint was contoured and adapted to the reset cast. In order that the splint could be transferred from the cast to similar tooth positions in the mouth, identifying markings were outlined on the cast. In the region of the left first incisor a vertical half-round tube was soldered to the splint, while in the region of the left first premolar a horizontal oval tube was soldered to the splint. To join these attachments a 14-gauge half-round post was soldered mesially to an 18-gauge all-round wire terminating in a threaded oval post (end of Angle arch with threaded end and nut). The mesial end was shaped similarly to the other intermaxillary spurs on the Winter splint. A spring was soldered to the midsection close to the post to lock incisally over the vertical end of the half-round tube on the right side of the splint section, thus adding to the rigidity of the attachment. An adjustable nut fitted over the distal threaded oval end. This end engaged the horizontal oval tube of the left splint section. This section, a practical jackscrew, was removable and could be shortened or lengthened as desired. To make the section effective, the corresponding intervening section of the Winter splint, which until now maintained the desired contour in relationship to the arch, was cut off, thereby forming a three-sectional splint. These divided parts, when reassembled, formed a stable continuous mandibular splint, conforming to the reset mandibular segments (Fig. 2, *B*). In selecting teeth to be banded to improve the stability of the end sections, loose or mobile teeth were avoided. Bands with spurs were constructed and contoured for the left second premolar and second molar, the right first incisor, canine, and the second molar.

The maxillary and mandibular bands were cemented on the teeth. The maxillary splint was wired to each and every tooth using a double thickness of 0.009 inch stainless wire (Fig. 3, *B*). Before inserting the left and right sections of the mandibular splint, markings

corresponding to those on the casts were transferred to the mandibular teeth. Guided by these indicators the end sections were securely ligated into place (Fig. 3, *C*). The middle removable section was retained to be inserted at the time of operation for refracturing (Fig. 3, *D* and *E*).

The patient was hospitalized and prepared for operation. The following day her mandible was operated on under a general anesthetic. An external incision about 3 inches in length was made through the integument along the lower border of the mandible in the region corresponding to the original fracture. Then the muscles were carefully reflected using blunt dissection until the bone was exposed. The periosteum of the bone was raised. Overriding bone could be discerned by the unusual thickness of the mandible. Diagonally across the inferior border a very slight line of demarcation was detected. On this line with a sharp chisel and mallet the bone was fractured. The tissues were replaced and sutured into position. Then the midsection of the mandibular splint was set into place, the spring locked over the half-round tube of the splint right end section, the adjustable nut set flat against the oval tube of the left end section. Intermaxillary rubbers were inserted (Fig. 4). The following morning her teeth bit into occlusion, thus moving the fractured segments into proper relationship to the maxilla. At this time the nut was set firmly against the oval tube.

It is interesting to report that intermaxillary rubbers of stethoscope tubing were used instead of intermaxillary wires. The rubbers were easily inserted. They had the advantage of ease of control of intermaxillary force in the direction desired. Should nausea or vomiting require the removal of intermaxillary force to permit the patient to open her mouth, it could be done without disturbing the splints. The continuous force of rubbers also had a singular advantage of overcoming muscular spasms present in fractures.

During the following six weeks a nourishing liquid or semiliquid diet, i.e., fruit juices, egg-nogs, purée of vegetables, milk, etc., was prescribed and oral hygienic measures observed. At the end of this period the intermaxillary rubbers and both maxillary and mandibular splints were removed.

A bony union resulted with a favorable alignment of the fractured segments (Fig. 3, *F*) and a good functional occlusion (Fig. 1, *D* and 2, *C*). The excursions of the mandible were free and favorable. Her speech difficulty was entirely eliminated. There was a notable improvement in her facial appearance (Fig. 1, *C*) as well as her psychological well-being. In fact, all the relief the patient sought seemed achieved.

#### SUMMARY OF CASE

Patient sustained multiple jaw fractures in an automobile accident. Although treated immediately, she was disappointed and discouraged with the result since there was a malunion of the mandibular fracture in the region of the mental foramen. It caused a severe malocclusion with a disfiguring facial deformity which interfered with mastication, limited mandibular excursions, and hindered enunciation.

The malocclusion could not be corrected by orthodontic methods nor by surgical methods alone. It required the utilization of the resources of the surgeon and orthodontist cooperating and coordinating their preoperative preparation, operative and postoperative care. To correct this deformity it was necessary (1) to anticipate the new alignment of the mandibular segments, (2) to design and construct a maxillary and mandibular (three-sectional) splint, (3) to reinforce maxillary and mandibular anchorages by cementing bands with spurs on key teeth, (4) to insert the maxillary splint and the end sections of the mandibular splint prior to the osteotomy, (5) to refracture surgically the mandible in the line of the original fracture and to insert the midsection of the splint at the time of operation, and (6) to use intermaxillary rubbers instead of wires for intermaxillary fixation.

The combined surgical and orthodontic procedure (1) brought about a favorable psychological readjustment in the patient, (2) improved the esthetic appearance by correcting the malunion, (3) restored a functional occlusion, (4) improved mastication, and (5) restored enunciation.

140 EAST 54TH STREET.

57 WEST 57TH STREET.

## DUAL FUNCTIONAL BITE

### A CASE REPORT

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THE patient, a boy 14 years of age, was of normal stature but his appearance suggested an early arrestment (Figs. 1 and 2). This was due to a dull depressed look to be found in his face, occasioned by a marked underdevelopment of the maxilla and a constantly drooping lower jaw. Clinical examination showed that the patient could find no normal position of rest in articulation (Fig. 3). The mandible would swing from side to side until it found one of the two positions that would permit him to achieve mastication but not comfort. In either position there was occlusion on only one side; the mandibular teeth of the other side were entirely lingual in their relation to the maxilla. The upper jaw was wider than normal in the molar and premolar region, whereas the lower jaw in the same region was slightly collapsed. In occlusion the chin was deflected to one side or the other, dependent upon the choice of the patient to close to either side. Because of the type of the malocclusion there was a cross-bite, with the attendant constant trauma of the crossing anterior teeth. It was a mouth that was in urgent need of orthodontic interference for functional, as well as esthetic, reasons.

The patient came of a normal middle-class family whose history showed nothing to indicate that there might have been any hereditary or prenatal influence upon this case of malocclusion. The child was delivered with instruments but there was no indication of injury at birth or at any time thereafter. During his childhood he had suffered none of the familiar children diseases which occasionally interfere with normal development, and had been bothered only by allergies of rose fever and hay fever with a mild sinusitis. His tonsils and adenoids were removed at the age of 4 years. He remained a mouth breather, however, until the orthodontic treatment had been completed.

Treatment was instituted in January, 1942, in the Orthodontic Clinic of the Hospital for Joint Diseases. Radiographs, photographs, and casts of both jaws were taken. A profile head radiograph was taken and this showed a well-formed skull and apparently normal jaw relationships when at rest (Fig. 4). Unfortunately a registration of the condylar positions were not recorded. The radiographic examination of the teeth showed them to be normal with a few well-constructed fillings. Since there were no temperomandibular pictures available, the case was approached from a purely clinical aspect.

From the Orthodontic Service, Hospital for Joint Diseases, New York, N. Y.  
Presented before the New York Society of Orthodontists, Nov. 14, 1944.





Fig. 1.



Fig. 2.

A.

B.

C.

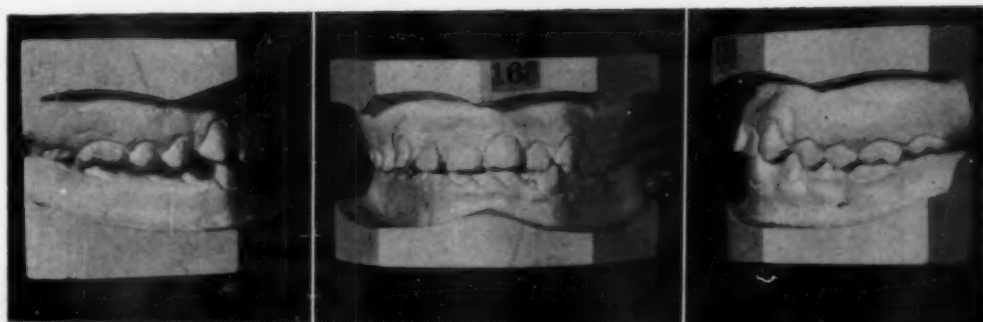
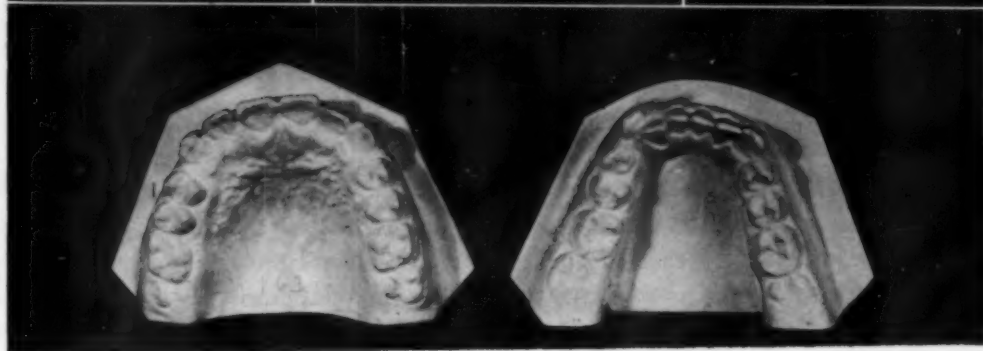


Fig. 3.



D.

It was necessary from the beginning to ascertain which of the two bites that the patient was able to assume was nearer to the normal clinically, so that it was efficient, comfortable, and in accordance with the facial harmony and cranial anatomy. We then had to determine if that bite could be utilized as a basis for the course of treatment we had contemplated. Careful examination of the patient during mastication and in both centric positions indicated that the position of the mandible when it was directed to the left appeared to be the most satisfactory and this recommended its choice. There was less chin deflection and a slightly better median line relationship.

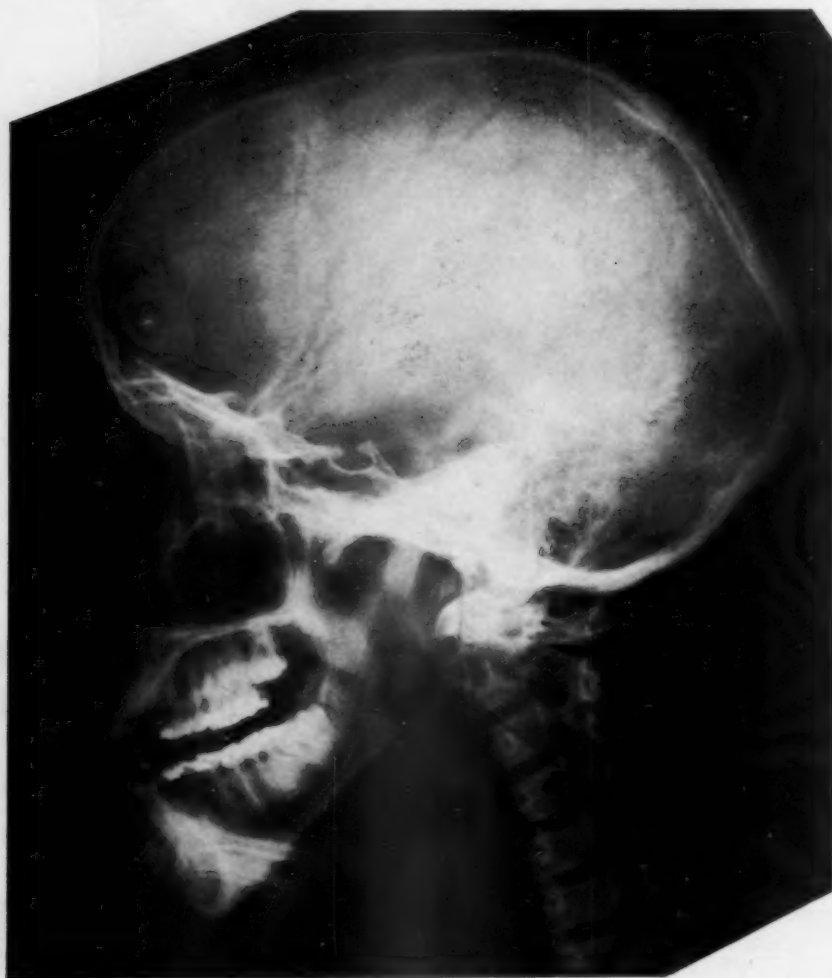


Fig. 4.

Having accepted this as a basis for operation, an upper vulcanite bite plate was constructed (Fig. 5). It was built so as to disengage the anterior teeth and thus eliminate the trauma that resulted from the cross-bite. There were added to the anterior retaining wire two buccal arms about 2 cm. in length directed posteriorly, which were carried back behind the second molars and imbedded in the vulcanite. To the buccal arm on the right side there had been attached two T bars so that force could be applied to the premolars that would drive them

lingually until they engaged the lower antagonizing teeth. As the maxillary premolars were moved lingually, the bite plate was festooned on its lingual contacting perimeter to allow for further movement as the teeth engaged the outlines of the bite plate. The molars involved were assisted in realizing correct buccolingual relations by the application of cross elastics.

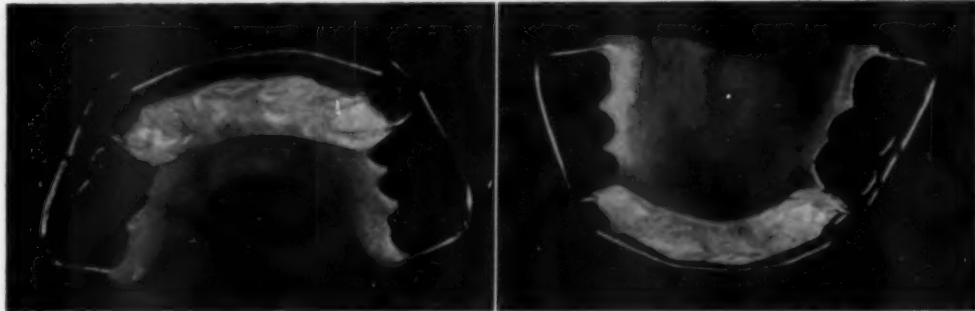
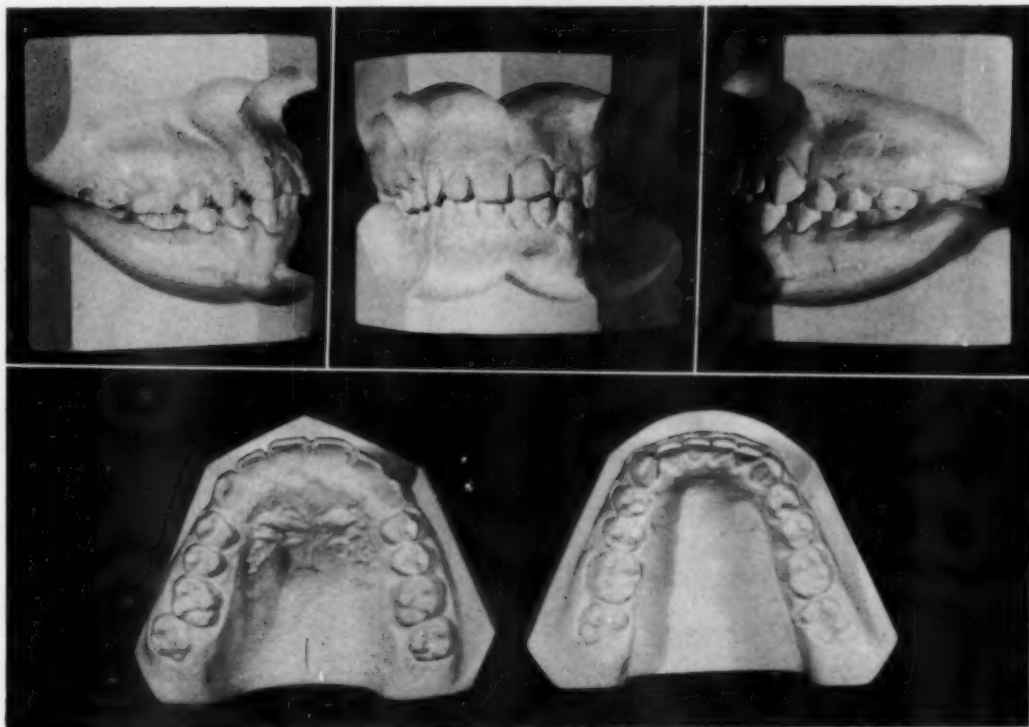


Fig. 5.

A.

B.

C.



D.

Fig. 6

In the mandible, lingual appliance was inserted at the same time from the first molar on one side to the first molar on the other, and, by means of auxiliary springs of 0.020 gauge, pressure was applied to the teeth on the right side. These were moved buccally simultaneously to meet the uppers which were being moved lingually.

Within a year and with a minimum of discomfort for the patient, the teeth had been brought to such a normal functional relation that there was occlusion on both sides. Having ascertained that the patient could tolerate the changes and that the shifting bite had been eliminated, the treatment was changed to the use of a modified edgewise appliance. All the teeth were measured and charted. From these measurements there was constructed a predetermined arch form. All the teeth were then banded with 0.003 by 0.125 inch bands to which had been soldered a modified edgewise bracket and staples for rotations, if necessary. The molars were banded with 0.006 by 0.180 inch band material and to



Fig. 7.



Fig. 8.

these molar bands were attached buccal tubes to receive a 0.022 by 0.028 inch wire. Regulating arch wires formed from the predetermined arch form were ligated to the teeth. The case was started with a 0.014 inch stainless steel wire, and then as the case progressed, the succeeding 0.016 and 0.018 inch wires were used, until finally a 0.022 by 0.028 inch precious metal arch was used to finish the case. During this time the patient was seen every four weeks, when the old arches were removed and new arches employed.



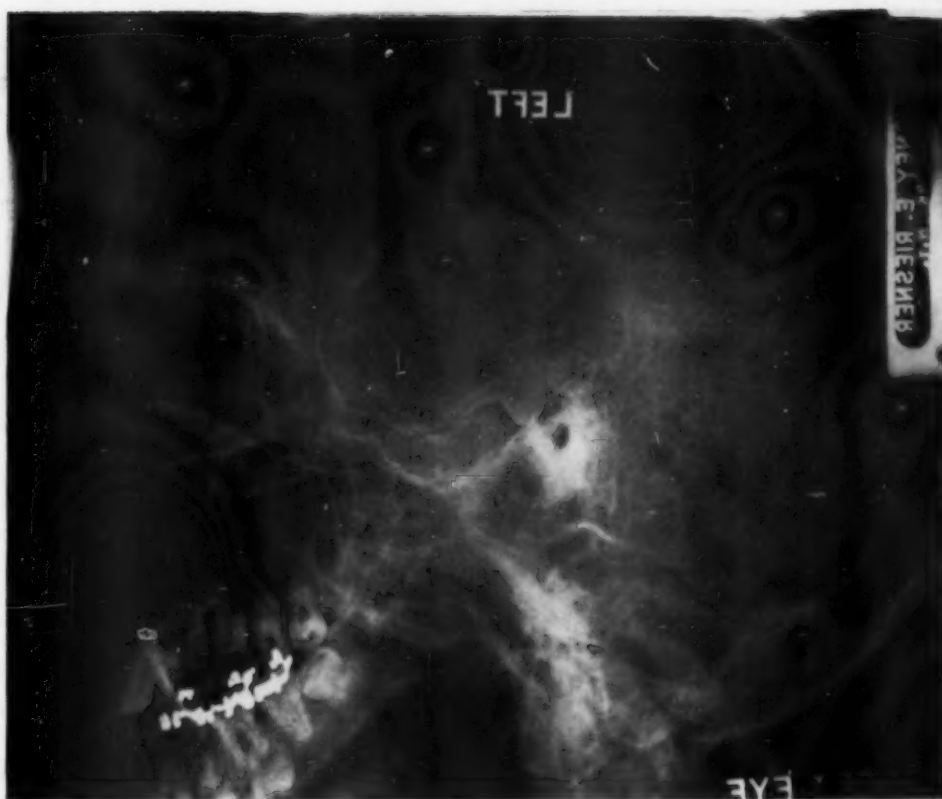


Fig. 9.

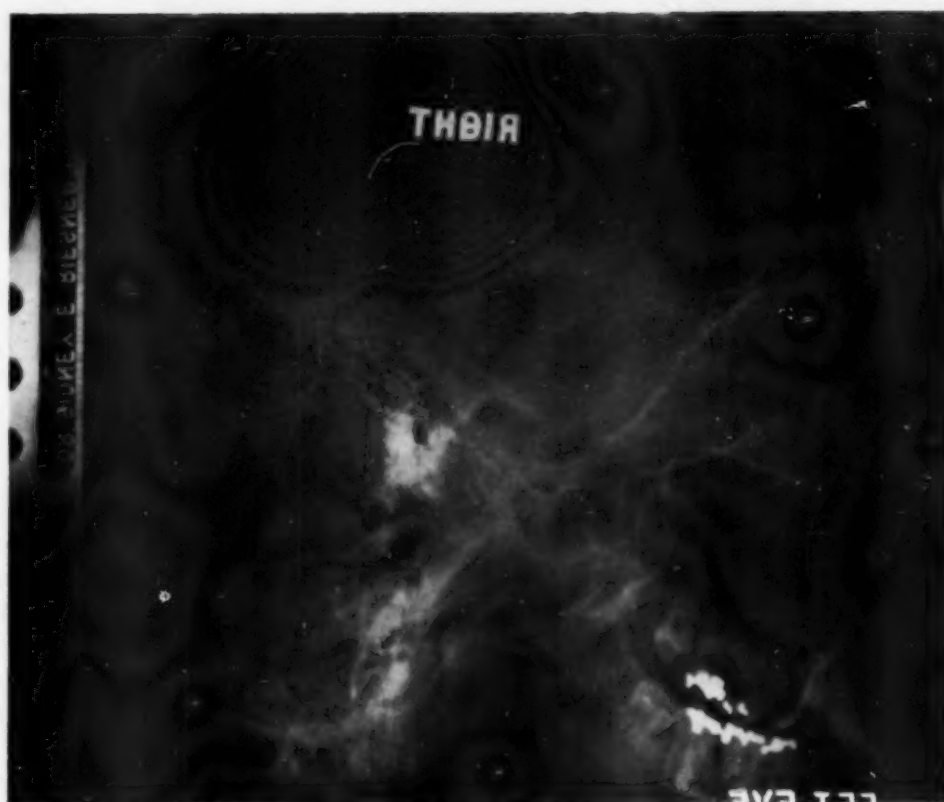


Fig. 10.

After twelve months the teeth were in good functional occlusion and the patient perfectly comfortable and happy. Fig. 6 shows casts of the completed case. After removing the appliances, the teeth appeared so well interdigitated that it was decided to eliminate the use of retainers. Stability of the dentures bore out the confidence of this, for, aside from a slight settling, the teeth have been self-retaining.

Esthetically the result is pleasant, and although there is a slight deflection of the chin to the left side, it is not objectionable (Fig. 7). The profile shows a marked development of the maxilla and a better facial contour (Fig. 8). There is an added alertness in his appearance and a complete loss of the instability of the mandible and the look that attends a patient suffering with enlarged adenoids.

It is quite evident that it would have been almost impossible to introduce treatment in this case had we attempted to select an intermediate position of the mandible as the basis from which to start. It is also interesting to note that practically all the movements in this case were accomplished by the use of intra-maxillary force.

Posttreatment roentgenograms of the temporomandibular joints, as shown in Figs. 9 and 10,\* revealed an interesting relation that was different in both joints. The left side exhibited a position mildly posterior and superior to the normal, while the right side showed an inferior and backward relation. While the lack of earlier registrations affords no means of comparison, it is interesting to note the accommodation the condyles have taken in response to the driving force of occlusal cusp guidance. Since the force exercised was essentially one of intramaxillary origin, it is significant that similar changes occur in our daily treatments of even far greater degree than these registered, because of the employment of intermaxillary elastics in different types of therapy.

Just what will happen to these joints in the future will be an interesting feature to watch. Where the readjustment will take place, and to what extent, will be pertinent and important considerations in future treatments.

8 WEST 40TH STREET

\*The Hospital is indebted to Dr. Sidney Riesner for the radiographs of the temporomandibular joints.

## SUPERNUMERARY TOOTH IN A CHILD OF TWELVE MONTHS

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**S**UPERNUMERARY teeth may occur in either deciduous or permanent dentition,<sup>1-3</sup> and are recognized as a direct cause of disturbed eruption and malocclusion.<sup>4, 5</sup> Although supplemental teeth are common, the appearance of such a tooth producing harmful effects in a child of 12 months seemed unusually interesting.



A.

B.

Fig. 1.—A, Labial aspect of supernumerary tooth. B, Mesial aspect of supernumerary tooth.

### HISTORY

S. D., a girl, aged 12 months, had been delivered normally at term, was apparently healthy in all respects, and had experienced no metabolic upsets or diseases prior to dental examination. Parents and grandparents were free of dental or other anomalies. First evidence of dental abnormality was delayed eruption and malpositioning of the maxillary right deciduous lateral incisor tooth observed at time of examination. The dentition had followed a normal course of eruption except for the tooth mentioned. Mandibular central and lateral incisors had erupted during the sixth and seventh months; their maxillary counterparts, except for the right lateral incisor, during the seventh and eighth months. In the twelfth month a tooth resembling an atypical deciduous canine (Fig. 1) erupted into the maxillary right canine region, forming a  $\frac{1}{4}$  inch diastema with the right central incisor. A radiograph (Fig. 2, A) revealed that the caninelike tooth was supernumerary and that the deciduous right lateral incisor was deflected from its normal course of eruption.

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The child was observed for a month, at the end of which it was decided to remove the anomalous tooth to prevent further aberration of the normal dentition. The operation was performed on Nov. 6, 1943, using divinyl ether (2 c.c.) by the open drop method without premedication. It was uneventful and recovery was rapid. The right lateral incisor began to erupt two weeks after the extraction. A radiograph (Fig. 2, *B*) of the maxillary right lateral incisor region, made at 20 months of age, indicates a normally positioned lateral incisor and developing succedaneous tooth bud.

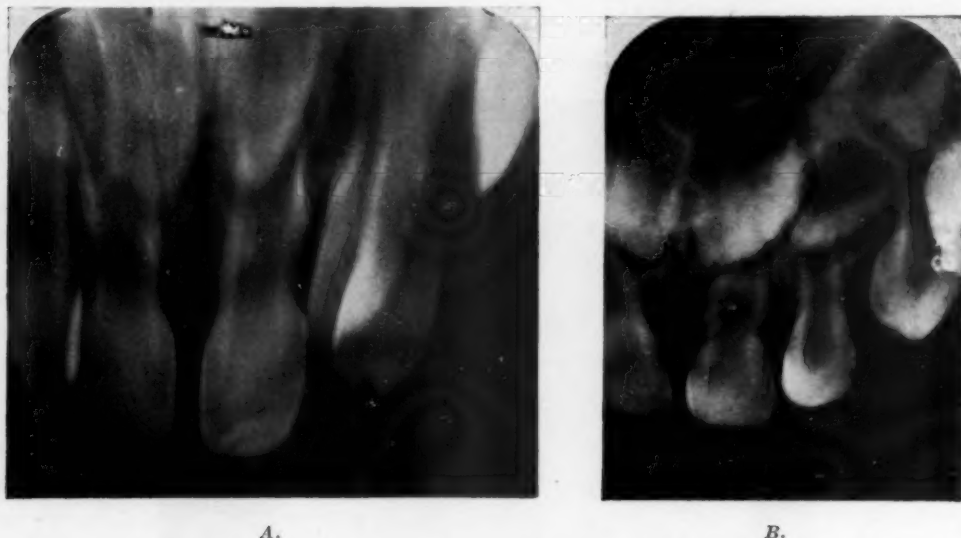


Fig. 2.—Radiographs of region of maxillary right lateral incisor tooth: *A*, Preoperative, visualizing supernumerary tooth superimposed on malposed deciduous lateral incisor. *B*, Seven months postoperative, showing lateral incisor erupted in normal position.

#### DISCUSSION

The extracted tooth (Fig. 1) is apparently a rarity among supernumerary teeth.<sup>6</sup> The progress of calcification of the root would indicate that the tooth developed simultaneously with the deciduous right lateral incisor. Although the genesis of supernumerary teeth is not clearly understood, that they develop by abnormal proliferation of the dental lamina is generally accepted.<sup>7</sup> This case illustrates the truth of the concept that early diagnosis and removal of supernumerary teeth is the most satisfactory method of preventing their untoward effects in the normal dentition.

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## ENAMEL HYPOPLASIA AND ITS PROBABLE RELATION TO ORAL DISEASE

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**H**YPOPLASIA of dental enamel has received a great deal of attention over a long period of time. Its role in the etiology of many oral conditions, however, has not been given due consideration. This is true especially of the relationship of hypoplasia of the dental enamel to many periodontal dyscrasias.

Hypoplasia of the dental enamel presents various clinical characteristics. Some of these forms are definitely more harmful than others to the oral health of the affected individual. It should be kept in mind that hypoplastic enamel is not a disease in itself but the result of a disease or diseases. Consequently, the causative agent of hypoplasia should be considered as the real etiological factor of the sequelae to hypoplastic teeth. But when we consider the countless pathologic conditions to which hypoplasia is attributed, the fact remains that these causative factors themselves have in most cases completely receded when the disturbances initiated by the presence of hypoplastic tooth forms take place.

In the following discussion we shall consider tooth hypoplasia as an independent entity in regard to the conditions that arise due to the weakness and deformities of hypoplastic teeth. We shall first consider separately the different types and forms of hypoplastic enamel and their influence on oral disease. The most interesting types to be studied are the indentations in the incisal edge of incisors and canines, the hypoplastic enamel in the occlusal surface of molars, and a whole series of dental deformations, hypoplastic in type, that have been named according to their form and location. Among them are the following: "Erosion," first applied by Fauchard, because of the resemblance to the erosion or corrosion that can be produced by acid acting on the enamel surface; "depression," as termed by Bunon; "tooth in honey-comb" (Tomes); "stony enamel" (Salter); "ondulated enamel" (Walkoff); "graded enamel" (Bethune), etc. Each one of these names actually indicates only one of the various aspects in which hypoplasia presents itself. The name "hypoplasia" was proposed by Zsigmondy (1893) and is today the term most accepted in America.

### HYPOPLASIA AT THE INCISAL EDGE OF TEETH

Incisors with an indentation or notch at the incisal third have been frequently wrongly designated by many authors as "Hutchinson" teeth. The fact remains, according to Jonathan Hutchinson (1828-1912), who first described syphilitic hypoplasia and gave its pathologic meaning, that the above-mentioned term should be used exclusively to denote the maxillary permanent central incisors which present a crescent-shaped depression of the incisal edge. This does not mean that the crescent-shaped cut is observed only in the max-

illary central incisors. It can be observed also in the mandibular incisors, maxillary lateral incisors, and maxillary and mandibular canines.

It must be pointed out that teeth with hypoplasia at the incisal border do not show any notching at the time of eruption but only a noticeable defective enamel in this region, more marked in the middle than in the mesial or distal ends of the incisal margin. Later, due to masticatory stress, this hypoplastic zone is worn away so that an indentation of varying depth is formed, depending on the duration and intensity of the causative forces of enamel hypoplasia. The notch may appear as a shallow deformation in the incisal edge or as a deeper indentation involving the whole depth of the incisal third and more rarely the middle third. As the time passes, these teeth lose their typical notch as the weak and defectively formed enamel which frames it is reduced by continuous masticatory action. In this manner in the adult stage these teeth present a straight or almost straight incisal edge.

Most often Hutchinson's teeth are ill shaped. Among the usual deformations are the "barrel tooth" and the "screw-driver tooth." In the first case the greatest circumference of the tooth is not located at the incisal edge but at its medium third; in the second form it is located at the gingival third.

To complete this description, it may be said that, besides the defective types described above, a particular type of malposition is frequently observed in Hutchinson's teeth (maxillary central incisors). They appear separated by a space sometimes quite large, showing in addition an inclination of their vertical axis converging toward the median line.

Correct form and position of teeth are considered today as factors of paramount importance in tooth and gingival health. When teeth are affected by hypoplasia, they can prove to be definitely harmful to the health of the oral tissues. Abnormal anatomy, as observed in the "barrel tooth" and the "screw-driver tooth," produces a tendency to drive food into the proximal spaces of these teeth through occlusal pressure (vertical impaction) as well as by tongue and lip action (horizontal impaction). Proximal contact is disturbed and the health of the interdental papillae suffers.

The food impaction described can prove to be most harmful to the dental and periodontal tissues and generally brings about increased deposits of calculus, materia alba, mucinous plaques, etc., which complicate the already abnormal conditions.

Contrary to other types of hypoplasia, caries does not usually affect the crescent-shaped area of hypoplastic enamel because of the self-cleansing area in which it is located. Proximal caries is not infrequent, however, because of the malformations that are usually found in these teeth.

The weakened tooth structure in the incisal area, which, as was pointed out, is easily worn away, could create in the case of the lower incisors a condition of nonocclusion with their antagonists. This is known to cause a corresponding weakening in the periodontal membrane. In the case of the maxillary teeth, due to the changes in the inclined planes of the teeth, an exaggeration of the protrusive movements may take place which may cause trauma of some or all of the posterior teeth because of disruption of cuspal interrelation. Ill effects may also be expected on the periodontal structures

as well as in the region of the temporomandibular articulation as a result of these changes in tooth relation.

#### HYPOPLASIA ON THE OCCLUSAL SURFACE OF MOLARS

Teeth with hypoplasia of the occlusal surface, commonly known also as "mulberry molars," are frequently observed. Basically, this lesion is similar to the one described in the notched incisors. The difference lies in the distribution of the hypoplasia as it is modified by the shape of the tooth itself. In the case of the molars, the lesion is found widely spread on the occlusal surface, while in the incisors, due to the anatomy of the incisal edge, hypoplasia appears in the form previously mentioned.

Molars with hypoplasia, instead of their normally white, smooth, and vaulted cusps, present an eroded surface which is pitted, rough, irregular, and has sharp or hornlike projections, or as Zinsser says, "volcanic-like elevations." The hypoplasia ordinarily affects all of the occlusal area and frequently also part of the lateral walls. On the other hand, the lesion may be limited to the extremities of the cusps. The extent of the lesion actually depends upon the duration of the etiological factor. In cases where the pathologic forces are intensively exerted over a relatively longer period of time, two-thirds or three-fourths of the molars may remain unaffected, while the remaining coronal portions appear as prominent projections, small, twisted, irregular in shape, and considerably reduced in size. The foregoing is the description of a young tooth. Since the defective area is structurally weak, it is shaved off more or less rapidly in function so that it is later replaced by a deep, yellowish-gray depression surrounded by an areola of white enamel.

This type of anomaly is symmetrical and occurs bilaterally, so when it is found in the maxillary right first molar, for instance, it will also be found in the maxillary left molar, and very probably also in the mandibular first molars. The damaging effects of such conditions over the masticatory apparatus, and hence to the general health, are self-evident. Considering that the teeth most commonly affected by this type of hypoplasia are the permanent first molars, it can be easily understood that they cannot carry on their important function of "key of the occlusion" as they are normally expected to do.

What is worse, because of their weak construction, they readily become subject to devastating caries of the occlusal surface. As a result, when these teeth are not treated in time, they are usually extracted, producing complete disorganization of the main forces of occlusion by shifting of the adjacent teeth, loss of proximal contact, nonocclusion, as well as traumatic occlusion, increasing the susceptibility to decay in the remaining teeth. All of these factors are of primary importance in the etiology of periodontal disease.

Even under favorable conditions, when these affected teeth through one cause or another escape the ravages of decay, their morbidity is still high.

Due to the peculiar shape of hypoplastic molars, food is forced into the interdental spaces, producing chronic gingivitis with subsequent pocket formation. This condition is found especially in young people because of the presence of the irregularly shaped cusps (plunge cusps) already described, which increase the wedging of food. In this period the occlusal surface of



hypoplastic teeth is an ideal place for the lodgment of food deposits, and it is difficult to eradicate them from it. Later, since the occlusal surface of the tooth easily wears off, the defective plunge cusps disappear, but interdental food impaction persists. Furthermore, the resulting flat surface produces an unsatisfactory masticatory environment as the areas of teeth contact are considerably increased producing exaggerated muscular activity in chewing. This later condition tends to overload the periodontal fibers, making them more prone to periodontal disease. Contrarily, it may be stated that persons having teeth affected in this manner will instinctively reduce their masticatory force. When this is the case, however, they subconsciously select softer food, with the resulting increase of susceptibility to decay as well as creating a weak and loosely woven alveolar structure, which is more prone to periodontal disease.

When the occlusal surface of hypoplastic teeth becomes flat, lateral pressure which normally occurs in young intercusping teeth does not exist. Hence, there is no stimulation of the periodontal fibers in the lateral excursions of the mandible. This condition has been known to promote atrophy and recession of the gingival tissue.

Closure of the bite with its sequelae of temporomandibular disturbances and muscular disbalance usually follows either the loss of molars or their rapid and abnormal wear. Needless to say, nutritional disturbances are prone to follow insufficient masticatory function.

We shall now briefly describe two other groups or clinical types: "hypoplasia in fossa" and "hypoplasia in furrow."

#### HYPOPLASIA IN FOSSA

This condition is characterized by the appearance in the buccal surface of the crown of one or more pits or fossae of more or less perfect circular shape. When numerous, these almost always show a typical distribution. The pits which occur near the free border of the tooth assume a horizontal distribution; the others adopt irregular positions. These depressions, always pigmented, generally show increase in depth as they approach the occlusal limits of the crown. These depressions in the enamel occur more frequently in the incisors than in the molars.

#### HYPOPLASIA IN FURROW

This condition constitutes one of the most commonly occurring types of hypoplasia. The furrows always assume a horizontal distribution. In microscopic sections, they are found to run parallel to the lines of Retzius.

The extent of the furrows varies considerably. They may affect only one-third or one-half of the buccal surface (benign type) or in more severe cases they may embrace the tooth circumferentially, strangling it at an even distance from the incisal edge of the crown. Sometimes teeth present several superimposed furrows that give them a peculiar appearance. They are called by French authors "*dentis étage*," "*en grandin*," or "*en escalier*," because of their resemblance to gradings of an amphitheater or staircase. Bethune calls them "*graded teeth*," because they measure quite accurately the alternate periods in which the etiological factor recurs and remits, causing alternate periods of normal calcification and failure of calcification.



The ill effects of these latter two types of anomalies are not as apparent as in the cases previously presented here. Of course, there is the fact of weakened structure and its proneness to decay with its sequelae.

There is a psychological factor, however, that contributes to this condition a harmful concomitant. In the case of multiple furrows of the anterior teeth, a rather unsightly appearance is found. Hence there exists the probability that the individual affected will not consider these teeth worth the proper care and will not bother with brushing, massaging, etc. This attitude will render the whole mouth more susceptible not only to decay but also to periodontal disease. In addition, psychosomatic factors (accused by some schools of thought of producing periodontal dyscrasies) may play a role in this condition, principally in young women to whom personal appearance is of such paramount importance.

#### HYPOPLASIA IN TEMPORARY TEETH

Hypoplasia of the temporary dentition, considered by many authors to be quite rare, is indeed a familiar sight to the pedodontist and dentists rendering service in public clinics, where children of low-income families are examined in great numbers.

The impression that hypoplasia of the dental enamel in deciduous teeth is a rarity is probably due to the fact that until recent years children's dentistry was considered unimportant by dentists as well as parents. This unfortunate opinion still exists in many countries where public dental clinics for children are practically unknown.

The types of hypoplasia observed in the deciduous dentition are exactly the same as those described above as occurring in the permanent teeth, modified of course by the differences in anatomy and strength of children's teeth. Hypoplastic conditions are seen in children of all economic levels but most frequently affects children in families of limited means and the indigents. Frequently all of the deciduous teeth are affected, a condition which rarely if ever occurs in the permanent dentition. The sequelae are obvious; it is a well-recognized fact in dentistry that the deciduous dentition influences the health of the permanent teeth.

As hypoplasia produces a premature loss of the deciduous teeth, it can eventually give rise to all kinds of malocclusions and malpositions of the permanent teeth, with their traumatogenic as well as infective sequences; and, going even further, psychological disturbances due to the unesthetic facial appearances are sequelae of these conditions.

The overbite that results in severe cases of hypoplasia may leave its imprint over the delicate and moldable bones of the child, disturbing the normal relationship of bone, muscles, and ligaments in the temporomandibular articulation, that eventually will be prone to disarthrosis or subarthrosis with its sequelae of deafness, tinnitus, difficulty of speech, headaches, neuritis, etc. This overbite takes place not only in cases of premature loss of temporary teeth but even when they are treated on time. The usual procedure to treat this condition is elimination of any food-retentive irregularity on the teeth by grinding away, and the precipitation of silver nitrate on the hypoplastic area, due to the danger involved in constructing retentive cavities because of

pulp involvement as well as for economical reasons. Onlays and inlays for proper restoration of height, occlusion, and contour are not used in most public clinics and are rejected in most cases as "too expensive" by parents who bring their children to the private dentist.

The attempt has been made here to show the need for proper protective measures in individuals affected with extensive enamel hypoplasia. These measures should not only attempt to conserve the teeth but also to restore proper function through adequate height and width as well as proper anatomic outline and proximal contact. In doing so, countless harmful conditions affecting the teeth, occlusion, and periodontium will be prevented.

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Max E. Ernst, Secretary, American Association of Orthodontists, 1250 Lowry Medical Arts Bldg., St. Paul, Minn.



## Department of Orthodontic Abstracts and Reviews

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Edited by

DR. J. A. SALZMANN, NEW YORK CITY

All communications concerning further information about abstracted material and the acceptance of articles or books for consideration in this department should be addressed to Dr. J. A. Salzmann, 654 Madison Avenue, New York City

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### **Clinical and Roentgenographic Study of Effects of Hormonal Therapy on Bone Growth.** By Rita S. Finkler, N. J. Furst, and M. Klein, *Radiology* 43: 346, October, 1944.

An investigation by the authors at Beth Israel Hospital, New York, for the past eight years shows the influence of endocrine therapy in 81 children, 18 of whom were treated with thyroid substance, 26 with anterior pituitary growth extract, 19 with chorionic gonadotropin, and 18 with testosterone. Thyroid therapy tended to improve bone density and epiphyseal union. At the conclusion of therapy the bone density was normal in all children, but in five the delay in epiphyseal union persisted. There was an improvement in growth rate, physical development, and mental alertness. Best results were obtained in children with thyroid deficiency. Therapy was followed with anterior pituitary growth extract in the majority of the 26 patients. Chorionic gonadotropin therapy stimulated bone growth in the longitudinal axis but did not accelerate epiphyseal union, bone maturation, or density in the majority of the cases. Seventeen children responded by an increase in growth rate; two children maintained their original rate of growth. All children showed an improvement in genital development, muscular tone, mental alertness, and social adjustment. There was also a loss of weight in the majority of the obese children. Testosterone therapy showed a tendency to accelerate skeletal growth in length to a somewhat greater degree than chorionic gonadotropin. Clinically, 16 children showed an increase in growth. All children showed improvement in genital development, muscular tone, and self-assurance. Following endocrine therapy, with improvement in growth and genital and muscular development, there was a tendency to improvement in mental and emotional stability.

### **Fluorine and Fractures.** Editorial, *J. A. M. A.* 127: 399-400, Feb. 17, 1945.

Twenty years ago attention was directed to the discovery of fluoride in domestic water supplies as the causative factor in dental fluorosis, or mottled enamel. In the intervening period intensive investigation has been made of the physiologic chemistry of this element with respect to its influence on the skeleton, its metabolic effects and its relation to dental caries. In view of the observation that fluorine in the drinking water is correlated with a significant decrease in dental caries, suggestions have been made that fluoride be added to city water supplies in areas where this element is absent. However, the

need for caution has been repeatedly expressed, as several less desirable effects of ingested fluoride have been noted. One of these is the decrease in breaking strength of bones when fluoride is present in the diet.

A recent study of the influence of fluoride in domestic water on height, weight, and fracture experience affords important data on this question. Taking advantage of the vast quantity of statistics accumulated in connection with the induction of young men into the Armed Forces, McClure compared the data for height and weight of 2,529 selectees, some from areas where the drinking water contains fluorine and some from areas whose water was free from fluorine or below the known level of dental fluorosis. In like manner 1,458 high school boys were studied. In addition, statements were obtained from each subject regarding bone fractures such as are encountered in various sports. After correlating the data on height, body weight, and fractures with the broad spread of concentrations of fluorine in the water drunk by the subjects, the conclusion was reached that fluorine in the water has no effect on height or weight and appears to exert no serious impairment in skeletal performance such as might be manifest in broken bones.

The urine of a large proportion of the same subjects was examined for fluoride concentration. In localities where the water was free of fluorine, the average concentration in the urine is 0.3 to 0.5 part per million. With increasing amounts of fluorine in the water there occurred proportional increases in the urine. It appears from the foregoing observations that normal renal function is a safeguard against suspected toxic bone fluorosis in adult human subjects even in regions where the potable waters contain appreciable amounts of fluorine.

## News and Notes

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### **The Red Cross—An Ally in the Campaign for Better Teeth**

The war against tooth decay, especially in growing children, is an old campaign with the dental profession. But it becomes more evident every day that the battle cannot be won by any single group in the nation. The job calls for a concerted offensive by all agencies dedicated to the improved health of the people. Whether the dentists know it or not, one staunch ally in the campaign for better teeth is the American Red Cross and, more particularly, its Nutrition Service program.

Many factors are involved in the cause and prevention of dental caries, but every dentist knows the importance of nutrition in the development and maintenance of sound teeth. Through its nutrition program, the American Red Cross is giving this vital information to the nation's homemakers and breadwinners and to the youth who will be the homemakers and breadwinners of the future.

The national nutrition program, organized at the beginning of the present war, is designed to help people utilize food resources wisely that they may yield the fullest possible health returns. Local chapters of the American Red Cross are cooperating toward this end with representatives of the Agricultural Extension Service, Public Health Department, Farm Security Administration, the schools, and with the county and city nutrition committees. Nutrition education is made available by Red Cross chapters—and there are 3,757 of them blanketing the nation—in either a standard 20-hour course, or a modified version, requiring 12 hours.

These courses teach the food essentials necessary to the health of every member of the family. Equally as important, they demonstrate how specific nutritional requirements can be met by wise selection and proper preparation of available foodstuffs. Realizing that many persons have neither the time nor the interest to enroll in classes, many Red Cross chapters have employed additional means of carrying nutrition information to the people. Information centers, demonstrations, exhibits, films, talks, radio and newspaper publicity have all been used extensively to broaden the educational program.

Since 1941 American Red Cross chapters have issued more than one-half million certificates to persons satisfactorily completing nutrition courses. Over 90,000 of these were issued during 1944, and at the same time, other activities were reaching more than 350,000 persons. Certificates issued in Missouri total over 13,939. There are 230 instructors in the state authorized to teach Red Cross nutrition courses.

If these beginnings seem small, it must be remembered that changing food habits of many generations standing is a slow business. However, public interest in foods and nutrition has shown a marked increase during the past few years following the advent of the war. With nutrition courses being made available in more high schools and colleges every year and with the American Red Cross and other agencies in the community carrying nutrition information to adult groups, this progress is certain to continue.

It is by now an accepted fact that improved diet cannot only decrease the prevalence of dental caries, but can give some protection against the further development of tooth decay already present. The importance of adequate minerals and vitamins in the diet of the pregnant mother if her child is to develop sound teeth is also commonly accepted. While these may be truisms to every dentist, unfortunately he is often not consulted until repair work is needed. At that point, it is a little late to give information about diets and their relation to the development of teeth.

Here is where the Red Cross and other interested agencies prove valuable. Through its nutrition program, the Red Cross attempts to tell the people what the dentists would like to tell them if they always had the chance—that “food makes a difference.” They tell them the importance of minerals, vitamins, and other food nutrients in the development and main-

tenance of strong bodies and sound teeth. They tell them how to select and prepare foods; how to serve appetizing meals using the basic foods; how to provide adequate diets on a limited budget and how to get the most from ration points.

With a more widespread knowledge of nutrition and the practical application of such knowledge, it is reasonable to expect that real progress will be made in the prevention of tooth decay among growing children.

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### **Bedside Dentistry in Army Hospitals**

A portable dental unit is being used in certain Army hospitals to assure bedridden patients more complete dental care and speedy convalescence. Plans are now under way to standardize this unit for all Army general hospitals here. According to the Office of The Surgeon General this "dentist's office on wheels" carries equipment for a wide variety of dental operations from simple dental prophylaxis to treating fractured jaws and making complete new dentures.

A recent report on the portable dental unit at Kennedy General Hospital, Memphis, Tennessee, lists 363 dental treatments given in one month to bed patients there.

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### **Columbia University, Graduates in Orthodontics, 1944 and 1945**

Juan Diaz Zayas-Bazan, D.D.S., Havana, 1943  
Morris Matthew Berry, D.D.S., Temple, 1931  
Edwin Alvin Brown, D.D.S., Dalhousie (Canada), 1941  
Ulysses Erdreich, D.D.S., Columbia, 1941  
Bertram Emanuel Gerzog, D.D.S., Columbia, 1942  
David Marshall, D.D.S., Maryland, 1942  
David Mossberg, D.D.S., Pennsylvania, 1934  
John Raymond Santeramo, D.D.S., Maryland, 1941  
Paul Christian Sexauer, D.D.S., Columbia, 1940  
Joseph A. Sheldon, D.D.S., Louisville, 1943

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### **Refresher Training for Army Dentists**

Refresher professional training has been authorized for officers of the Army Dental Corps who have been assigned to other than professional duties or who have been limited to one phase of dentistry for a year or more. Priority will be given those who have served overseas.

This training, which is voluntary, is not intended for dental officers desiring to specialize, but provides about three months' rotation in all phases of general service. Requests must be initiated by the individual officers concerned and submitted through channels.

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### **Note of Interest**

Dr. M. Albert Munblatt announces the removal of his office to 57 West Fifty-Seventh Street, New York 19, N. Y. Suite 1402. Plaza 3-9436. Practice limited to orthodontics.



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\*The Journal will publish the names of the president and secretary-treasurer of foreign orthodontic societies if the information is sent direct to the editor, 8022 Forsythe, St. Louis 5, Mo., U. S. A.

JANUARY, 1945

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including

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## Original Articles

### CHRONIC DESQUAMATIVE GINGIVITIS

#### A REPORT OF TWELVE CASES

DANIEL E. ZISKIN, D.D.S., AND EDWARD V. ZEGARELLI, B.S., M.S., D.D.S.,  
NEW YORK, N. Y.

**D**URING the past decade, we studied a number of cases of chronic desquamative gingivitis. This report covers observations on twelve of these patients (two men and ten women) attending the Columbia University-Presbyterian Hospital Medical Center, other clinics, or referred from private practices. Four were in the third decade of life, two in the fourth, four in the fifth, and two in the seventh. The common complaint was red, raw, painful, hemorrhagic gums. All the cases were representative and presented histories with certain features in common. In most instances, anti-Vincent's or conservative periodontal treatment had been resorted to. Systemic therapy, including liver (parenteral and oral) and vitamins, especially B and C, had been tried. In some, radiation therapy and autovaccines had been used. All these forms of treatment proved unsuccessful.

The disease had been present for varying periods from a few months to several years at the time of our initial examination (See Table I).

Chronic desquamative gingivitis is not common. It is found in both men and women, more frequently in the latter. It is a disease of adulthood, occurring usually during middle life, although a few of our cases were between 22 and 30 years of age.

Tomes,<sup>1</sup> in 1859, described a gingival lesion characterized by a smooth, polished surface with acute intermittent pain. He noted that patients with such lesions were usually middle-aged women in whom menstruation was becoming irregular or had ceased altogether.

Goadby<sup>2</sup> reported a chronic lesion of the gingival mucous membranes which was bright red, injected, and accompanied by soreness.

Prinz<sup>3</sup> characterized desquamative gingivitis as an epithelial desquamation of the marginal gum tissue, the exposed connective tissue being deep bluish red in color and bleeding easily on the slightest provocation. He observed that the disease did not manifest itself in edentulous regions. He assumed that endocrine disturbances might play a role in its etiology since it occurred most frequently in middle-aged women in whom menstruation had become irregular or who were at the menopause.

Merritt<sup>4</sup> reported that the disease is more common than is generally believed and that in its milder and less typical forms it may be confused with chronic gingivitis. Cahn<sup>5</sup> also described the clinical picture of desquamative

From the School of Dental and Oral Surgery, Columbia University.

TABLE I  
TWELVE CASES OF DESQUAMATIVE GINGIVITIS

CASE	AGE IN YEARS	SEX	RACE	TYPE*	LOCATION†				DURATION	PAIN‡	PREVIOUS TREATMENT								TREATMENT				RESULTS§	COMMENT
					ALVEOLAR	AREOLAR	EDENTULOUS	PALATAL			APOXESIS	ANTI-VINCENT'S	VITAMINS	MOUTHWASHES	CAUTERIZATION	X-RAY	VACCINES	EXCISION	PAREN-TERAL		TOPICAL			
																			ANDROGENIC	ESTROGENIC	ANDROGENIC	ESTROGENIC		
1. J. S.	37	F	W	1, 2	✓	✓	✓	✓	8 yr.	++	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	++	Vitamin B therapy caused severe exacerbation of mouth lesions
2. G. W.	44	F	W	2	✓	✓	✓	✓	1 yr.	+	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	+++	Recurred two years following ces- sation of treatment
3. B. O.	29	F	W	1	✓	✓	✓	✓	3½ yr.	+	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	+	Inadequate treatment
4. R. W.	49	F	W	2	✓	✓	✓	✓	7 yr.	±	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	+++	No recurrence for past four years
5. J. M.	23	M	W	2	✓	✓	✓	✓	1 yr.	±	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	+++	Recurred after treatments were discontinued
6. A. L.	21	F	W	2	✓	✓	✓	✓	1 yr.	±	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	+++	Recurred in a mild form after cessation of treatment
7. F. S.	40	F	W	1, 2	✓	✓	✓	✓	3 yr.	++	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	+++	Rapid recurrences after cessation of treatment
8. G. E.	61	M	W	1, 2	✓	✓	✓	✓	5 yr.	++	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	+++	Still under mild treatment though gingivae appear normal clin- ically
9. C. P.	38	F	W	2	✓	✓	✓	✓	3 mo.	++	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	+++	Vitamin A in addition to estrogen was administered with good re- sults. Still under mild treat- ment
10. E. W.	67	F	N	1	✓	✓	✓	✓	1 yr.	±	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	+++	Occasional recurrence of single vesicles
11. H. W.	25	F	W	2	✓	✓	✓	✓	6 mo.	+	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	+++	Vitamin B complex caused necrot- ic papillae symptomatic of Vin- cent's infection
12. C. S.	43	F	W	2	✓	✓	✓	✓	2 mo.	±	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	+++	Treatment not completed

\*Type 1—Vesicular.

Type 2—Atrophic.

†Free gum margins uninvolved.

‡Degree of pain = ±, +, ++.

§Degree of improvement = +, ++, +++, +++++ indicates complete clinical healing.

gingivitis. He stated that the process originates in the stroma and that the loss of epithelium is caused by a nutritional disturbance of unexplained origin. Sorrin<sup>6</sup> stated that menopause does not play a role in the etiology of desquamative gingivitis. In his report of ten cases, "endocrine check-ups" revealed little information as to the cause.

A variety of methods for the treatment of chronic desquamative gingivitis has been advocated in the past with little or no success. Tomes<sup>1</sup> recommended the sulfate and carbonate salts of magnesium as aperients. Goadby<sup>2</sup> applied non-irritating lotions and prescribed antiscorbutic foods. Prinz<sup>3</sup> maintained that potassium chlorate followed by a course of arsenic was the best treatment. In the less serious cases, mild astringents like copper sulfate were used, while surgical excision was resorted to in the more serious ones. At the suggestion of the writers, Cahn<sup>5</sup> tried the estrogenic hormone on one case "over a long period of time, but without in any way altering the course of the disease." Sorrin<sup>6</sup> used many treatments, including surgery, dyes, silver nitrate, chromic acid, and arsphenamine, with no specific drug acting the same in every case. No improvement was noted in one case with the use of an estrogen salve (furnished by the writers); surgery afforded only temporary relief. Sorrin's most satisfactory results were obtained through use of beechwood creosote and iodine applied to the gums and covered with glycerite and tannic acid. Hirschfeld<sup>7</sup> described a case of desquamative gingivitis in a woman of 53 years, in which various local intense treatments over a period of six years failed to bring any marked improvement in the condition. Three years after treatment was discontinued, the condition had spontaneously cleared up. Hirschfeld reported six other patients with more or less similar conditions. All were women in whom the gingival symptoms began to manifest themselves at, or soon after, the beginning of menopause. In none of these did local measures bring about improvement.

In a series of experiments previously conducted to test the effects of the estrogenic hormones on the gingivae of monkeys<sup>8</sup> and on the gums of a large group of women with gynecologic disorders,<sup>9</sup> it was found that the estrogens stimulated epithelization as well as connective tissue formation. These agents were tried in the desquamative gingivitis group in the following manner.

#### METHODS

The details of each case are listed in the protocols, with a distinguishing classification described as "Type 1 and Type 2." A gingival biopsy (obtainable in all except one case), study models, full mouth x-rays, and medical histories were routine procedures. Physical examinations for the purpose of this study were available in nine cases.

Following the preliminary work-up, treatment was instituted. The sex hormones,\* in the form of an ointment, were applied topically to the gingivae. The male or the female hormones were used to correspond with the sex.

\*Oreton-M is an androgenic preparation in a tegin base (glyceryl monostearate) which contains 2 mg. of methyl testosterone per gram. Each tube referred to in this paper contained 25 Gm. of ointment or 50 mg. of methyl testosterone.

Progynon-DH is an estrogenic preparation in a tegin base which contains 1,000 R. U. (10,000 I. U.) of estradiol per gram. Each tube referred to in this paper contained 25 Gm. of ointment or 25,000 R. U. (250,000 I. U.) of estradiol per tube. Both the male and female sex hormones were supplied by the Schering Corporation, Bloomfield, New Jersey. Progynon-DH in tegin base is an experimental preparation which is not at present commercially available. Progynon-DH ointment as regularly supplied has a lanolin base, but the Oreton-M ointment is available in a tegin base.

The dosage of each application was roughly 4,000 R.U. for the women and about 6 or 7 mg. of methyl testosterone for men. A large portion was lost through drooling so that the actual adherent amount was unknown. Disturbances in the menstrual cycle occurred in two instances, whereupon succeeding doses were reduced in these cases. Otherwise, the amounts absorbed seemed insufficient to cause observable systemic reaction. The ointments were administered in two ways. One consisted of their application to the gums in a retaining appliance made by adapting a shellac baseplate to a plaster cast of the patient's jaws. Space was provided for the ointment. By this method the hormone was kept in contact with the gum tissues for a half hour to an hour at each sitting (three to five sittings per week). This procedure was supplemented by a home treatment consisting of application of the ointment to the gums with the finger. Patients were instructed to spread the salve on the gums and to avoid any form of rubbing or massage. It should be emphasized here that no other form of therapy except that described was used.

In a few mild cases, patients were permitted their accustomed toothbrushing. In the more severe cases, even toothbrushing was discontinued. Periodontal treatments, including prophylaxis, were not given until the disease was well under control. Rigid limitation of therapy was maintained in order to establish a more accurate means of evaluating the hormonal effect. (For control purposes, the ointment base alone was applied in a number of patients with a variety of conditions without effect.)

Instructions were given to avoid trauma to the gums. Intense hormone medication for two to three months was followed by gradually decreased doses. The total time of treatment varied from a few months to two years in most cases, and longer in some. (See Table I for summary of cases.)

#### PROTOCOLS

CASE 1.—J. S., a white woman, aged 37 years, married, with one child 9 years of age.

*Medical History.*—During the summer of 1931, the patient presented with a scaly patch on the left temple and the front and lateral neck regions which had been present for eight or nine years and produced intermittent episodes of intense itching. The diagnosis was eczema and treatment consisted of radiation to the areas for four weeks. The lesions showed improvement, and intermittent light x-ray treatments were continued over a period of three months. After this, there was a recurrence and a new series of radiation treatments was begun. At this time, mouth symptoms, which patient stated had been present for a few years, were also noted. During the next five years, patient attended the dermatology clinic at irregular intervals. The patient observed that the mouth lesions appeared at the same time as the more severe episodes of itching of the skin. Skin tests for allergy revealed sensitivity to fish. No other sensitivities were noted. Special diets were prescribed. Patient also noticed exacerbations during menstrual periods. Scratch tests showed a marked reaction to staphylococcus toxoid and a moderate one to streptococcus. All other tests were negative.

*Medical Examination.*—Medical examination at this time revealed that the skin lesions on the neck were still present. They were described as a



seborrheic type of eczema. Gynecologic examination was negative, as were other medical phases.

*Dental History.*—Patient complained of watery blisters appearing on the gums at intervals coinciding with the more aggravated episodes of itching of the skin. The blisters burst, leaving raw, painful ulcers for several days before healing. This oral condition had been present for about eight years before hormonal therapy was instituted. During this eight-year period, the patient received intensive routine periodontal treatment. The gingivae generally improved, but the desquamative lesions continued to recur unabated. Among the diagnoses made were herpes simplex, aphthous stomatitis, and Vincent's angina. In 1936, this patient was referred to us for hormonal therapy of the gums.

*Dental Examination.*—All teeth were present with the exception of the upper left cuspid and first and second premolars, which were lost as the result of an automobile accident. Several teeth contained metal fillings. No active caries was present. The patient was wearing a unilateral upper partial denture. Her occlusion was normal.

The lesions were located generally throughout the entire gingivae, both upper and lower. The marginal gingivae were swollen and cyanotic in color. There were areas which appeared deep red, raw, and hemorrhagic. Such areas were seen on the palate, upper left side, and on the areolar gingivae between upper left lateral incisor to first molar edentulous area and upper right cuspid to first molar region. The lower gingivae presented large vesicles on the areolar portion varying in location, sometimes on the lingual and at other times on the labial, which burst, leaving shallow, desquamated, painful areas. The vesicular lesions appeared most often about a week before the menstrual period, but their occurrence was not confined to this time. The vesicles were transitory, varying in location. The desquamated areas in the upper jaw were constant. Both the areolar and alveolar gingivae were involved but not the free gum margin.

*Diagnosis.*—Chronic desquamative gingivitis of the vesicular variety (Type 1).

*Treatment.*—May 22, 1936, to June 12, 1936, she received 10 c.c. of  $\alpha$ -estradiol benzoate intramuscularly. Each cubic centimeter contained 5,000 international units. The gingivae generally improved while the areas of desquamation were diminished in size. June 13, 1936, to July 7, 1936, no treatment was given. The lesions became worse. July 7, 1936, to July 17, 1936, four (1 c.c.) injections of the estrogen were given. July 17, 1936, to Sept. 25, 1936, the patient received no treatment. She stated that her mouth felt better during the summer months. Sept. 25, 1936, to March 2, 1937, thirty-five injections of estrogen were given at periodic intervals. Approximately 45,500 international units were administered from May, 1936, to March, 1937. The injections were discontinued at this time. No new ulcers appeared for ten months. The mouth was free of subjective symptoms. A small area of desquamation in the edentulous area, upper left lateral incisor to first molar, remained. The condition of the marginal gingivae also improved.

On Jan. 25, 1938, vesicles reappeared. These started one week before the last menstrual period and continued to form throughout the period. The

patient did not proceed with her treatment until May, when the topical method was begun.

May 20, 1938, to March 28, 1939, estrogen ointment was applied topically for thirty minutes once or twice a day. Desquamated areas improved materially. Patient spread the salve on her gums daily before retiring.

From March 28, 1939, to the present time, patient was supplied with the ointment at infrequent intervals. Her mouth was examined about twice a month. The oral lesions were found to be well controlled. They reappeared occasionally, in mild form.

Recently, on patient's own initiative, she began taking vitamin B complex. Approximately 100 capsules were ingested. There was an acute exacerbation of the mouth symptoms. Upon discontinuance of the B complex the symptoms subsided.

*Follow-Up Note.*—While the desquamative lesions in this case never completely disappeared, they were greatly improved. The subjective symptoms were generally alleviated and the disease greatly ameliorated.

CASE 2.—G. W., a white woman, aged 44 years.

*Complaint.*—Patient complained of sensitive, bleeding gingivae.

*Medical History.*—She had received treatment in dental and dermatological clinics for over a year. Therapy included baking soda, x-ray, liver extract injections, and vitamin A-B-D capsules. No improvement was noted. Vincent's smears were positive; mouth culture showed diphtheroids isolated anaerobically; *Streptococcus viridans* predominated on plate. Cultures and smears for monilia or fungus infection, blood smear, Wassermann, and repeat Wassermann were negative.

*Medical Examination.*—Medical examination at our direction was negative. The menstrual flow was moderate, lasting four days, with regular 28-day painless periods. There were no menopausal symptoms.

*Dental History.*—Patient had been treated with sodium perborate, zinc peroxide, 2 per cent chromic acid, apoxesis, and prophylaxis.

*Dental Examination.*—Maxillary and mandibular anterior gingivae were red, raw, and denuded of surface epithelium to the mucobuccal fold; posterior and edentulous areas were involved.

*Diagnosis.*—Chronic desquamative gingivitis of the atrophic variety (Type 2).

*Treatment.*—Estrogen ointment was applied topically thirty-four times in twelve weeks; following this period, less than 15,000 rat units were used in daily home applications over three weeks. At this time, July 14, 1938, the desquamated area on the maxillary anterior labial gingivae was reduced to 1 to 1½ millimeters. (Its original extent was 8 mm. from the margins to the mucobuccal fold.) There was a new mucous membrane, firm, adherent, continuous, and purplish in color. In the mandibular anterior region the desquamation was only about ½ mm. in width. The edentulous areas which originally were involved almost solidly were now entirely healed.

From July 14, 1938, to Nov. 8, 1939, a period of about sixteen months, the patient received 775,000 rat units of estrogen ointment applied topically. From November, 1939, until the present day, infrequent and sporadic treatment was

administered, depending upon exacerbation of the lesions. For a period of at least two and one-half years, the patient was entirely free of clinical signs and symptoms. Recently the lesions returned in mild form. After a few topical applications of the ointment, improvement was again noted. Therapy is still in progress.



Fig. 1.—Case 3. Type 1 (vesicular) desquamative gingivitis located over right central and lateral incisors. The lesion was confined to the upper anterior region.

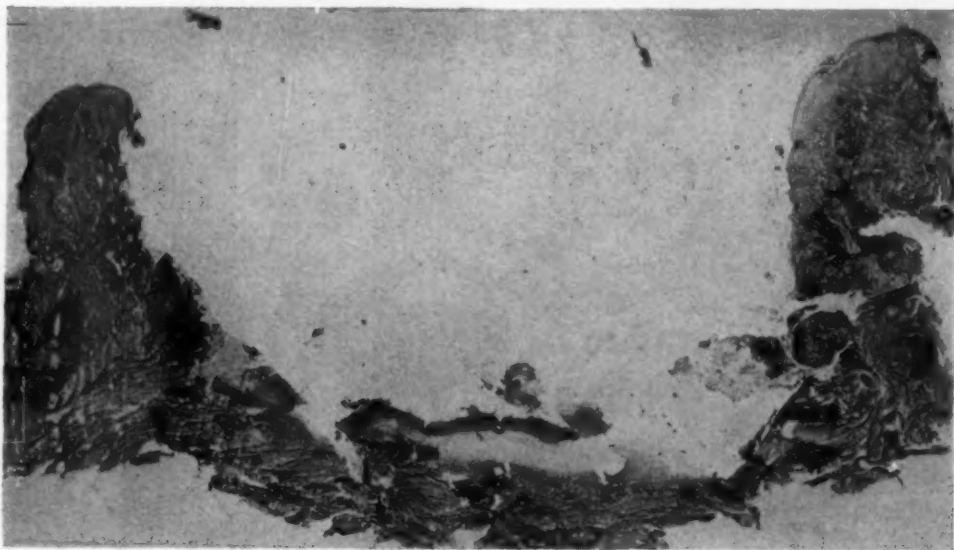


Fig. 2.—Case 3. Shows microscopic pathology of lesion seen in Fig. 1. The ulcer formed on rupture of the vesicle.  $\times 28$ .

CASE 3.—B. O'B., a white woman, aged 29 years.

*Medical History.*—Negative.

*Dental History.*—A desquamated lesion over the upper right central and lateral incisors was present for three and one-half years (Figs. 1 and 2). Vincent's smears were negative. Blood counts were negative. Subgingival curettage and conservative periodontal treatment proved ineffectual. Subsequent excision

of the lesion resulted in its prompt recurrence and spread to the left central incisor region.

*Dental Examination.*—There was present an inflamed, reddened, raw area on the labial alveolar and areolar gingivae over the upper right central and lateral incisors and the left central incisor (Figs. 1 and 2). The lesion was confined to this area. The remainder of the oral tissues appeared normal. One tooth was missing from each quadrant of the mouth. There were few fillings or carious teeth. Roentgenograms showed the alveolar bone to be normal.

*Diagnosis.*—Chronic desquamative gingivitis of the vesicular variety (Type 1).

*Treatment.*—Estrogen ointment was applied locally for one month with improvement noted. The patient did not return for further treatment.

CASE 4.—R. W., a white woman, single, aged 49 years.

*Complaint.*—Two years ago the patient first presented with a red and raw area on the mucous membrane over the upper left central and lateral incisors which bled on traumatization but was not painful.

*Medical History.*—Tonsillectomy at the age of 20 years; appendectomy at the age of 30 years. Fourteen years ago a diagnosis was made of migraine and albuminuria (cause unknown). Eight years ago, the patient was admitted to the hospital with acute bronchitis, asthma, pulmonary emphysema, generalized arteriosclerosis, and hypertension. Five years ago an x-ray of the heart showed a dilation and tortuosity of the aorta. The patient was under the care of several physicians. Ephedrine gave relief to the asthma; the headaches subsided with rest; the highest blood pressure recorded was 195/112. A year ago the basal metabolism was +23 per cent; a diagnosis was made of hyperthyroidism without goiter. Blood count and blood sugar were normal.

*Dental History.*—The desquamative lesion appeared seven years previous to our examination, with occasional remissions. Recurrences often coincided with the onset of menstruation. The area was treated at home for about a month with gentian violet, with slight improvement. Three years after appearance of the lesion it was excised. It recurred within six months, with no alleviation of symptoms.

*Dental Examination.*—Most of the teeth were present. The gingivae generally were firm and healthy in appearance. The lesion, about 2 cm. long and 3 mm. wide, was located in both the alveolar and areolar gingivae approximately 1 mm. above the free gum margin. It bled easily on being probed.

*Diagnosis.*—Chronic desquamative gingivitis of the atrophic variety (Type 2).

*Treatment.*—Fifty thousand units of estrogen ointment were spread on the gums by the patient over a period of one month, followed by 25,000 units over a period of several months. The symptoms of desquamation disappeared. Sporadic applications up to the present time were ordered as a preventive measure. There were no recurrences. The patient is still under observation.

CASE 5.—J. M., a white man, aged 23 years.

*Complaint.*—Patient presented with bright red, shiny, swollen, and spongy gums.





Fig. 3.—Case 5. Shows the gingivae and generalized desquamation. The lesion was also present in the anterior half of the hard palate.

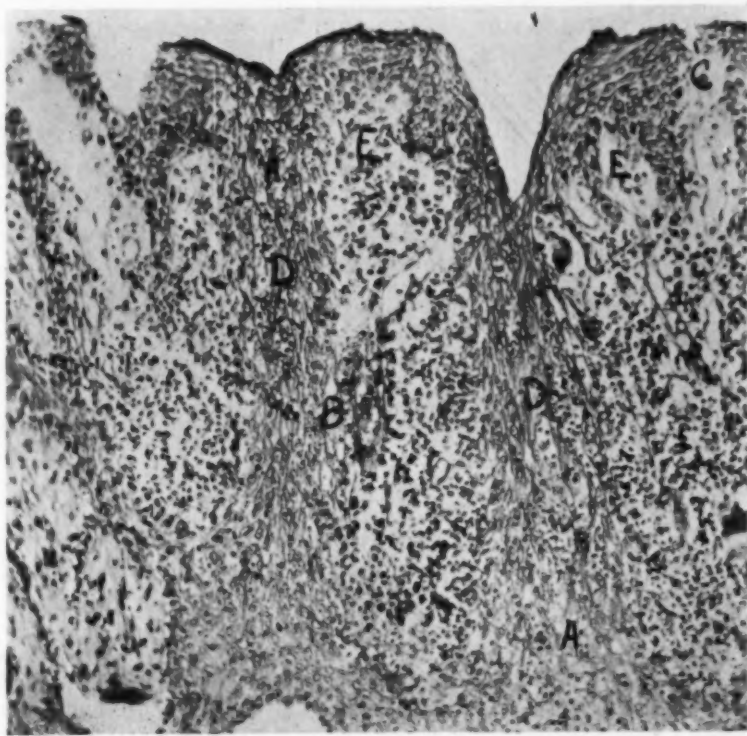


Fig. 4.—Case 5. Shows microscopic pathology of lesion seen in Fig. 3. Upper left No. 3 interdental papilla. Type 2 (atrophic) desquamative gingivitis. A, Hydropic degeneration of epithelium. B, Marked edema. C, Tears in epithelium as a result of the widespread edema. D, Round cell infiltration in epithelium. E, Edematous papillae of corium extending close to surface.  $\times 185$ .

*Medical History.*—Negative.

*Dental History.*—Patient received anti-Vincent's treatment at the hands of his dentist for approximately one year.

*Medical Examination.*—Physical examination revealed a slow pulse, dry hair and skin, and mental retardation suggestive of hypothyroidism. Basal metabolism test was not done.

*Dental Examination.*—Lesions were seen over the labial gingivae of the upper teeth from the right first premolar to the left first premolar, about 6.5 cm. in length and 10 mm. in width, extending from the crests of the papillae upward. The papillae were swollen and congested. A small lesion 5 by 5 mm. appeared on the buccal gingivae of the upper left second molar. The palatal mucosa presented a similar lesion from the upper right second premolar to the upper left first premolar, from the gingival margins across the anterior portion of the palate. Another small lesion, 5 mm. in width, in the lower left buccal gingivae, extended from the lower left second premolar to the first molar. A lesion about 1 cm. by 2 cm. was present on the labial gingiva from the lower right cuspid to the lower left cuspid. The affected areas were not painful and did not bleed on probing. Because of the swelling, the lesions appeared hypertrophic (Figs. 3 and 4). The remainder of the mucous membranes appeared normal.

*Diagnosis.*—Chronic desquamative gingivitis of the atrophic variety (Type 2).

*Treatment.*—Topical applications of a male sex hormone (testosterone) were administered. Nine applications, averaging over one hour each, were given in a period of 52 days. The clinical therapy was augmented by daily home applications. The patient then joined the Army and was not seen at the clinic for five months although the hormone ointment was sent to him. Upon his return, three more treatments were given, after which he again left the city. In all, approximately 700 mg. were applied.

The lesions showed signs of rapid healing. During the 52-day treatment period, most of the affected areas cleared up with the exception of those on the palate. The latter showed marked improvement. After a lapse of a year, the examining military dental officer reported a recurrence. We had no further contact with the patient.

CASE 6.—A. L., a white woman, single, aged 21 years.

*Complaint.*—Patient presented with brilliant red, raw lesions involving the buccal and labial gingivae of the upper and lower jaws. These lesions bled easily and were slightly sensitive.

*Medical History.*—Menstruation began at 15 years of age. Periods were irregular. Two years previously, the patient was told by a physician that she had a small goiter. On the patient's own initiative, iodized salt and spinach were added to her diet. She had gained 10 pounds in the past year.

*Dental History.*—Existent gingival condition was of one year's duration. Patient received no treatment.

*Medical examination* and accompanying history: Wassermann was negative; blood count was normal; basal metabolic rate was -3 per cent. Menses, last-

ing five days, occurred at six-week intervals. The flow was heavy, accompanied by backache and cramps on the first day. Headaches, nausea, and dizziness were relieved by self-induced vomiting. The latter symptoms had no time relationship to the menstrual period. The patient's physician diagnosed hypothyroidism. Thyroid medication was prescribed. Only 100 one-grain tablets were consumed.

*Dental Examination.*—The teeth and alveolar bone were in good condition; hygiene was fair with some calculus and sordes present. The interdental papillae generally were slightly cyanotic and swollen. There was some marginal gingivitis. On the upper right side there was a large area, raw, beefy-red in color, which bled easily on being probed (Fig. 5). This was the most prominent lesion, measuring 6 cm. by  $\frac{1}{2}$  cm. and extending from the midline to the second molar, beginning at the margins and progressing upward nearly to the mucobuccal fold. Most of the crests of the interdental papillae were not involved. Other areas of involvement were observed in the following locations: upper left lateral incisor, cuspid, second premolar, and first molar regions, and the lower interproximal papillae from lower right first molar to lower left cuspid (Figs. 6, 7, 8, and 9).



Fig. 5.—Case 6. Large lesion extends from upper left central to upper right first molar (dotted lines) area and involves both areolar and alveolar gingivae. There are also lesions visible in the lower anterior region. Pretreatment photograph.

*Diagnosis.*—Chronic desquamative gingivitis of the atrophic variety (Type 2).

*Treatment.*—Twenty-five topical applications of estrogen ointment were administered over a period of six weeks. The gingivae healed promptly. Therapy was suspended for four months. Some regression was noted. Treatments, augmented by daily home applications, were resumed for six weeks at weekly intervals. The gingivae again improved. Discontinuance of medication for five months resulted in a recurrence of the symptoms. The final series consisted of fifteen treatments at the chair, augmented by daily home applications, over a period of nine weeks. The patient was called in for follow-up one and one-half years later.

*Results.*—The healing of the desquamated areas was marked by a thin epithelial covering. This began as small skin grafts in small areas, later

coalescing to form a continuous layer. The new covering was thin at the outset, allowing the red color to show through. Upon superficial examination, the gingivae appeared unchanged. On closer inspection, the epithelial covering was easily detected (Fig. 10). As treatment progressed, the epithelial covering became thicker and the red color began to fade. On suspension of therapy, the color was intensified but did not approach the severity of the original condition. With each series of treatments, the appearance of the gums improved. When treatment was concluded, after about fourteen months, the gingivae appeared almost normal. They were pink in color with only a faint indication of the former desquamation. At the follow-up examination one and one-half years later, the gingivae were still free of desquamated areas except for a 2 mm. spot on the labial gingiva of the lower left central incisor.

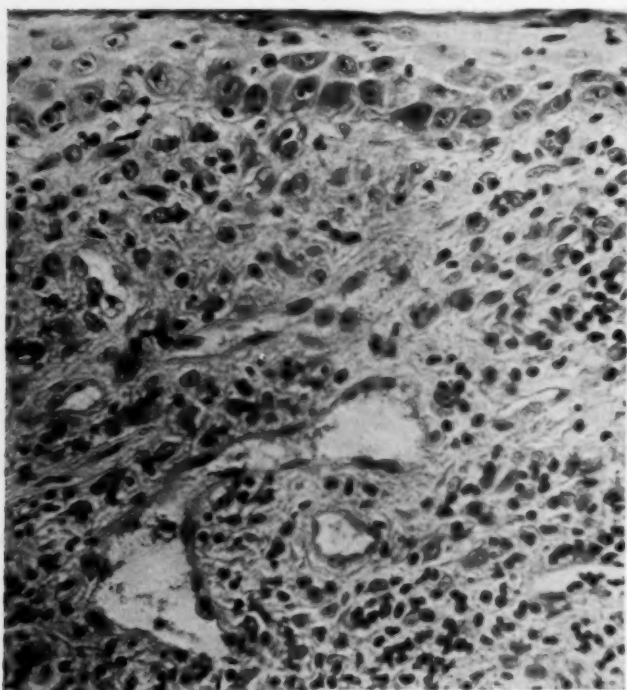


Fig. 6.

Fig. 6.—Case 6. Alveolar gingiva. Upper right No. 4 interdental papilla at crest. Shows marked edema and inflammatory infiltration into the epithelium.  $\times 490$ .

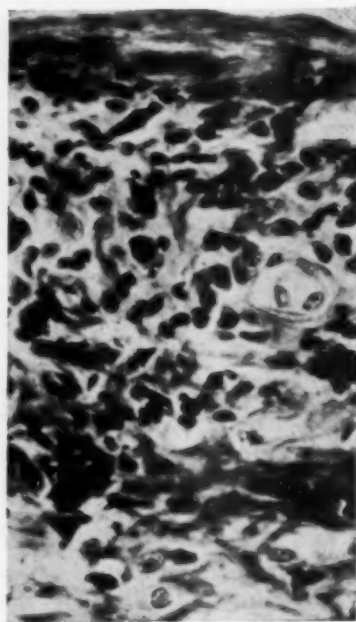


Fig. 7.

Fig. 7.—Case 6. Same tissue as in Fig. 6. Alveolar gingiva near its junction with areolar. Shows the absence of epithelium with the exception of the keratin layer which is seen covering the corium.  $\times 890$ .

CASE 7.—F. S., a white woman, married, aged 40 years.

*Complaint.*—Patient complained of painful, red areas on the oral mucous membranes which interfered with eating and sleeping.

*History.*—Appendectomy at 21 years of age, and cholecystectomy at 31 years of age. Diagnosis of gastric neurosis was made four years later. For the past five years menses had been irregular, occurring at varying intervals of two weeks to several months. Also, five years ago, a diagnosis was made of tuberculous peritonitis of the adhesive type. Two years ago, a gastric ulcer was dis-



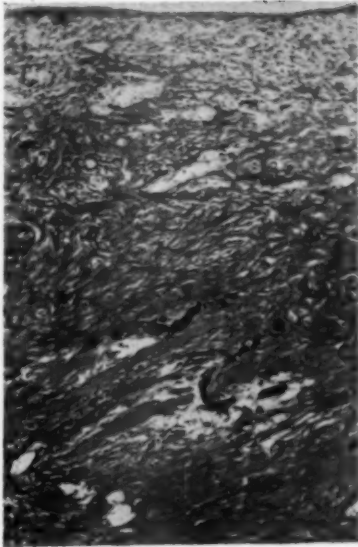


Fig. 8.

Fig. 8.—Case 6. Same tissue as in Figs. 6 and 7. Shows the corium denuded of its epithelial covering.  $\times 130$ .

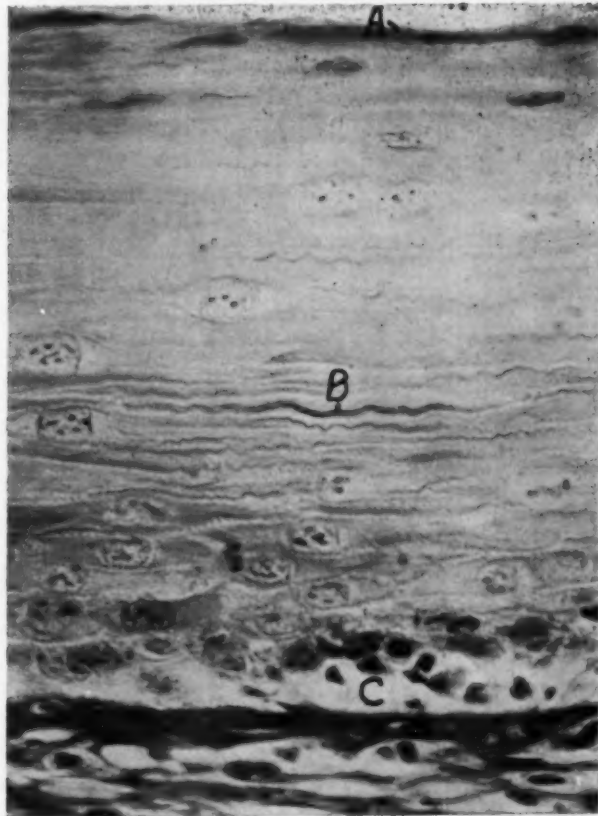


Fig. 9.

Fig. 9.—Case 6. Same tissue as in Figs. 6, 7, and 8. Areolar gingiva. *A*, Keratin on surface. *B*, Wavy lines showing compression of cytoplasm. *C*, Edema under basal cell layer causing flattening of rete pegs and atrophy of basal cells.  $\times 890$ .



Fig. 10.—Case 6. Same as in Fig. 5 after fifty-three days of treatment. Note the marked improvement in the clinical appearance of the lesion. Broken line indicates area where red of former desquamation is faintly visible.

covered. Recently the basal metabolism was -17 per cent and a fasting blood serum ascorbic acid determination was 0.4 mg. per cent.

Diagnosis of aphthous stomatitis was made three years ago in a nose and throat clinic where ultraviolet radiation therapy was given with negative results. The affection spread. Six months later the mouth lesions were diagnosed at the same clinic as chronic stomatitis. X-ray therapy (2,212 r.) produced no improvement. One per cent gentian violet was applied periodically; 10 per cent silver nitrate was used; a vaccine was prepared from the oral cultures and administered. There was no beneficial response to any of these forms of treatment. Surgical removal of the lesions resulted in their prompt recurrence.

*Dental Examination.*—The patient wore full dentures. The entire palatal mucosa was involved with a lesion which was soft, shiny, and of a deep red color. Several small ulcerated areas varying from 1 to 2 mm. to several centimeters in diameter were also present. The same type of lesion was seen in the upper buccal and lower buccal and lingual mucosa.

*Diagnosis.*—Chronic desquamative gingivitis of the vesicular and atrophic varieties (Types 1 and 2).

*Treatment.*—Estrogen ointment was applied, using the patient's dentures as retainers. Treatments were given twice daily, each session lasting one hour. Within two days, the patient noted a marked decrease in pain, and the lesions appeared less inflamed. After six weeks there was a complete cessation of pain, and the lesions were healed with the exception of a few small areas on the buccal surface of the upper right tuberosity and the mucosa of the hard palate. Self-application of the ointment followed at irregular intervals, or when there were signs of recurrences. Examination four months after beginning of treatments showed the mouth almost normal. The patient was not available for follow-up.

*Comment.*—The benefits with the application of the hormone were definite and the improvement noted was rapid. During lapses in home treatment recurrences were prompt.

CASE 8.—G. E., a white man, married, aged 61 years.

*Complaint.*—Patient was referred by a physician for diagnosis and treatment of sore, burning, raw, and bleeding gums. Patient stated mastication was painful.

*Medical History.*—Patient had a cholecystectomy three years previously. Injections of liver extract for a "run-down" condition, and high colonic irrigations were prescribed. Bilron capsules were taken. Iron, strychnine, and arsenic injections were given up to the time of our examination. Basal metabolic rate was -6 per cent.

*Dental History.*—The gum condition had been present for at least five years, during which time numerous forms of treatment were attempted. Among these were: apoxesis; oral prophylaxis; administration of vitamin C, B<sub>1</sub>, B<sub>2</sub>, and A in tablets, capsules, and injections; anti-Vincent's treatments using hydrogen peroxide and sodium perborate; and extraction of teeth in certain areas. Results were negative. At the time of our examination the patient stated that his gums were "as bad as ever."

*Dental Examination.*—The interdental papillae were blunted and marginal inflammation was present throughout the mouth. There were desquamated areas in the areolar and alveolar gingivae of the upper right and left sides, extending from the cuspid posteriorly to include the first molar area. The margins were not affected. Over the upper central incisors there were small patches which appeared hyperkeratinized. In the lower jaw, the gingivae had a violaceous color. Small areas of involvement were noted in the premolar area on the right and the cuspid and first molar edentulous areas on the left (Fig. 11).

Fig. 11.



Fig. 12.

Fig. 11.—Case 8. Type 2 desquamative gingivitis before treatment. Shows desquamated areas distal to the upper cuspids on the right and left sides (broken line).

Fig. 12.—Case 8. Same as in Fig. 11 after three and one-half months of treatment with the male sex hormone ointment applied topically. Desquamated areas are covered with a new layer of epithelium.

*Diagnosis.*—Chronic desquamative gingivitis of the vesicular and atrophic varieties (Types 1 and 2).

*Treatment.*—Topical applications of testosterone ointment were begun on Nov. 18, 1941, and continued for two months during which time the patient received 21 applications (10 mg. per application) with a total treatment time of eighteen hours. This was followed by self-application in accordance with the method used at the chair, about 50 mg. each week for another two months. Then

Fig. 13.

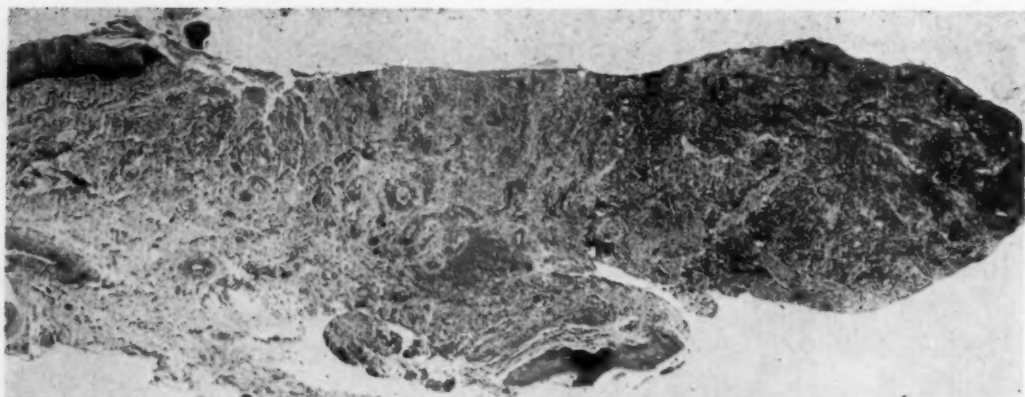


Fig. 14.



Fig. 15.

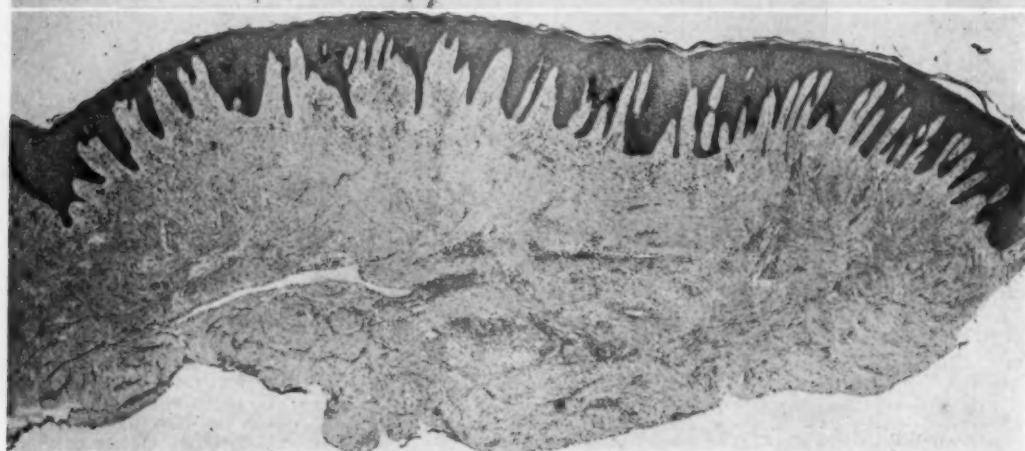


Fig. 13.—Case 8. Shows microscopic pathology of lesion seen in Fig. 11. Upper right No. 4 interdental papilla. Note complete absence of epithelium over most of the connective tissue. The intense inflammatory infiltration extends to the surface.  $\times 37$ .

Fig. 14.—Case 8. Same as in Fig. 13 after eleven months of topical application of the male sex hormone. Section was taken from the same area of gingiva as was shown in Fig. 13. Note the regeneration of the epithelial layer and the almost complete disappearance of the inflammatory exudate.  $\times 37$ .

Fig. 15.—Case 8. Same as in Figs. 13 and 14 after two years and seven months of treatment with the male sex hormone. Section was taken from the same area of gingiva as in Figs. 13 and 14. Here may be seen the healed gingiva as a result of treatment. A normal keratin layer is now present. The epithelium and connective tissue also appear normal although the number of pegs show an apparent increase and the corium is markedly increased in thickness.  $\times 37$ .



the home therapy was supplemented by twelve weekly intramuscular injections of 25 mg. each of testosterone propionate in sesame oil. The injections were repeated after a three-month lapse. They were discontinued for another three months and again repeated. Home applications were continued throughout, on five consecutive days each week.



Fig. 16.—Case 8. Higher power of Fig. 13 showing the intense inflammatory exudate and edema and the absence of epithelium.  $\times 120$ .

The results in this instance followed much the same pattern as did the other cases. Within three months, the patient noted marked symptomatic improvement, and within six months the lesions appeared healed clinically with cessation of all previous symptoms (Fig. 12). After eleven months, a second biopsy was taken. Findings were as follows:

The most striking feature of the second biopsy, when compared with the pretreatment section, was the regeneration of epithelium in the formerly desquamated areas. The epithelium was of normal thickness and took an even, clear, deep stain. Normal appearing nuclei were densely stained with hematoxylin. The cytoplasm contained occasional vacuoles. A new keratin layer was not seen, but in a small portion near the tip an area of parakeratosis was present. The epithelial pegs appeared somewhat flattened.

The connective tissue, in general, showed marked healing by the presence of dense bundles of normal-appearing collagen. Occasional granulomatous masses, composed principally of large round cells and plasma cells, were present, but greatly reduced in number and size. Inflammatory exudate was barely perceptible. Small capillaries were more prominently seen. In the subepithelial region there were zones which suggested characteristics of the connective tissue degeneration noted in the original biopsy, such as fragmentation of collagen, slight edema, inflammatory infiltration, and increased vascularity. These

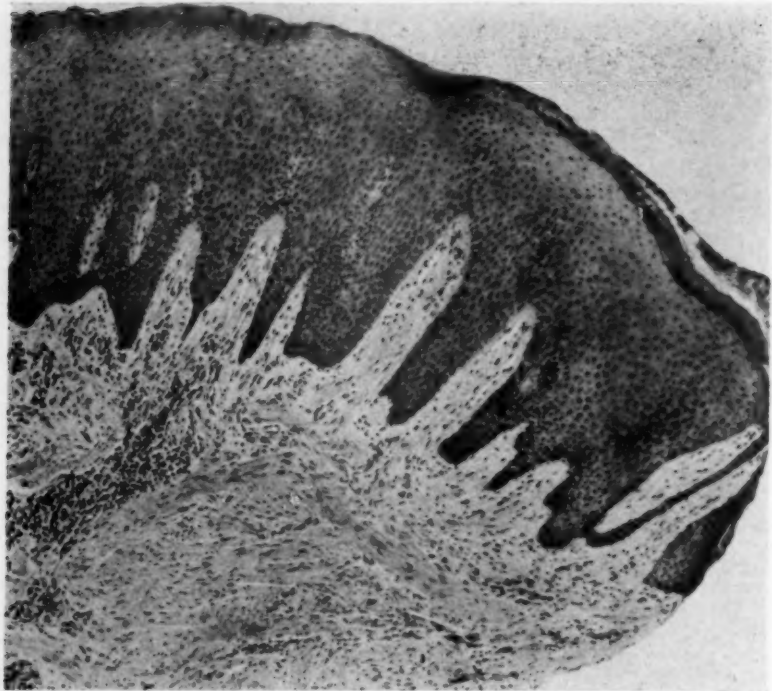


Fig. 17.—Case 8. Higher power of Fig. 14. Area is from a comparable location as that shown in Fig. 16. The new epithelial layer appears hyperplastic. The stratum corneum has not yet been restored with the exception of that seen in a small area at the tip. The inflammatory exudate has been greatly reduced but some perivascular infiltration is still present. The corium is dense and very cellular. Many fibroblasts are present.  $\times 84$ .

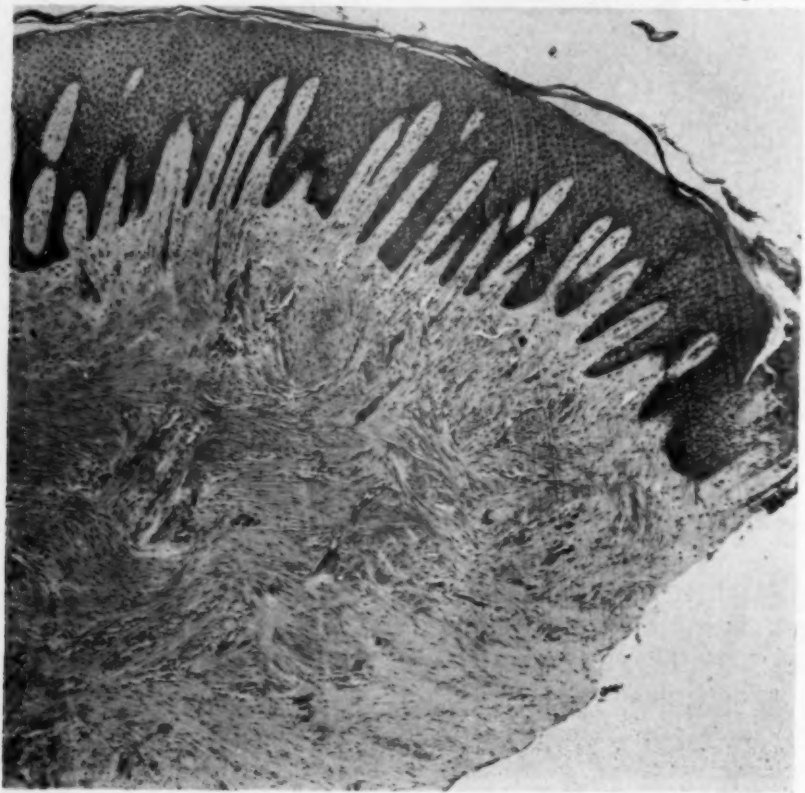


Fig. 18. Case 8. Higher power of Fig. 15. Compare with Figs. 16 and 17.  $\times 84$ .

areas were bounded by normal-appearing epithelium above and normal collagen below, indicating that the healing process was not yet completed. (Figs. 13, 14, 16, 17, and 19.)

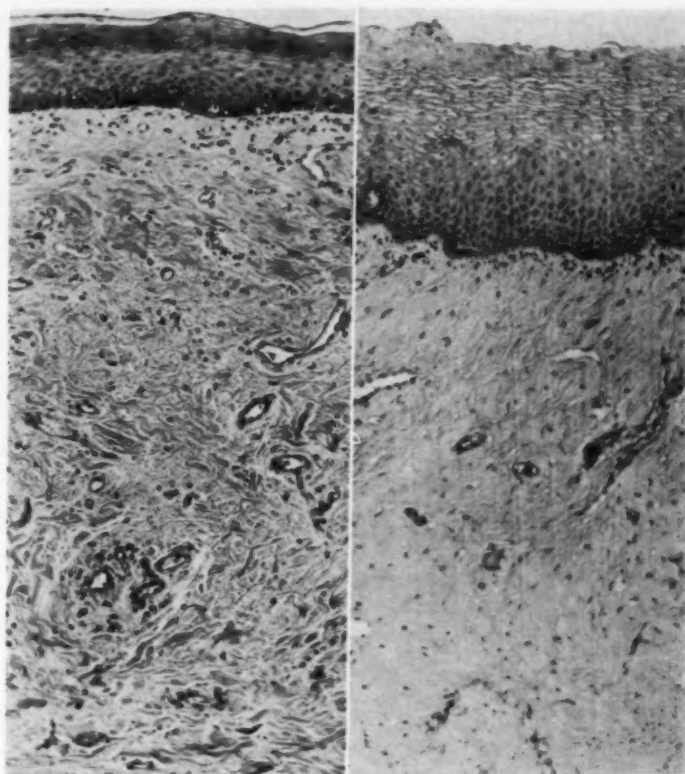


Fig. 19.

Fig. 20.

Fig. 19.—Case 8. Same tissue as in Fig. 14 showing the areolar gingiva. A slight amount of keratin and parakeratosis are seen on the surface. Slight perivascular infiltration is still present, particularly in the papillary layer.  $\times 127$ .

Fig. 20.—Case 8. Same tissue as Fig. 15 showing the areolar gingiva which appears more normal in that there is a disappearance of the keratin, a reappearance of intercellular bridges in the germinal layer and a denser corium practically free of inflammatory cells. Some parakeratosis still persists.  $\times 127$ .

Treatment was continued as before with the clinical appearance remaining normal. After about one and one-half years of treatment, toothbrushing was added to home hygienic care, which heretofore was confined to the use of a "denticator." It was noted at first that slight trauma induced "bleb" formation in isolated areas. As healing progressed, the gingivae became more resistant to irritations without vesicle formation. A third biopsy taken two years and seven months after the first shows the gingivae almost normal. A keratin layer was now present beneath which was a layer of parakeratosis of several cells in thickness. Vacuolization of epithelial cells, heretofore commonly seen, was conspicuously absent. Thickness of epithelial layer, staining, and the shape of the pegs were normal in appearance. The corium appeared completely healed and thicker than formerly. For the most part it was composed of dense bundles of collagen with a minimum number of inflammatory cells. The subepithelial zone, which was the site of earliest involvement and last to disappear with healing, was now apparently normal (Figs. 15, 18, and 20).



CASE 9.—C. P., a white woman, married, aged 38 years.

*Complaint.*—Stiffness and soreness of cheeks and tongue accompanied by a burning sensation of the mouth.

*Dental History.*—Three months prior to visiting our clinic, the patient became aware of an inflamed area on her gum over the upper left central and lateral incisors. On her own initiative, she used various mouthwashes, with no relief. Vitamin B complex had been taken during the last forty-five days of this period and the teeth had been brushed with a salt solution. No improvement was noted. On our instruction, the vitamin medication and toothbrushing were discontinued. A rubber finger massage was substituted. The lichen planus and desquamative gingivitis lesions remained unchanged.

*Dental Examination.*—Several posterior teeth were missing. The upper right lateral incisor was replaced by a fixed bridge. An upper right and a lower left premolar were devitalized. There were a number of amalgam restorations. Areas of desquamations were present in the gingivae, in the upper right central and lateral incisor, cuspid, and first molar regions; in the upper left central and lateral incisor, cuspid, and first premolar areas; and in the lower right first premolar, lower left first and second premolar, first and second molar areas. These lesions appeared raw, intensely inflamed, and bled easily. They were painful, irritated by many foods, rendering mastication difficult.

Under the tongue, on each side of the midline, was an inflamed area with a grayish membrane. An erythematous lesion 1 cm. in diameter was seen in the pharynx.

On the inner surfaces of both cheeks, the mucobuccal folds, hard and soft palates, and lateral borders of the tongue were lichen planus lesions with the typical violaceous color, mosaic lacy pattern, and depressed circular areas of bright red color.

Several aphthous ulcers were present on the mucous membranes in the region of the mucobuccal fold, floor of the mouth, and on the vermilion border of the lower lip.

*Diagnosis.*—Chronic desquamative gingivitis of the atrophic variety (Type 2), and lichen planus of the mucous membranes.

*Treatment.*—Estradiol benzoate ointment was applied at the chair once a week for twenty-five consecutive weeks (about 75,000 R.U.), augmented by daily home applications of the ointment at least once a day, usually before retiring. One tube per week (25,000 R.U.) was used in self-application.

After four months, the desquamative gingivitis showed symptomatic and clinical improvement. The reddened centers of the lichen planus lesions appeared paler.

Next, 25,000 units of vitamin A in oil, to be taken daily by mouth, were prescribed. Two weeks later, the dosage was increased to 50,000 units daily. This amount was augmented four months later by 100,000 U.S.P. units of vitamin A distillate in sesame oil injected intramuscularly once a week.



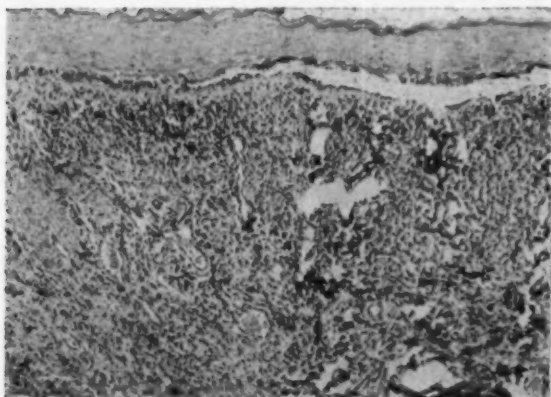


Fig. 21.—Case 9. Type 2 desquamative gingivitis. Upper left No. 1 interdental papilla. Before treatment with the estrogenic hormone. Shows blunting of epithelial pegs; subepithelial edema and stripping; marked edema and inflammatory exudate in corium.  $\times 92$ .

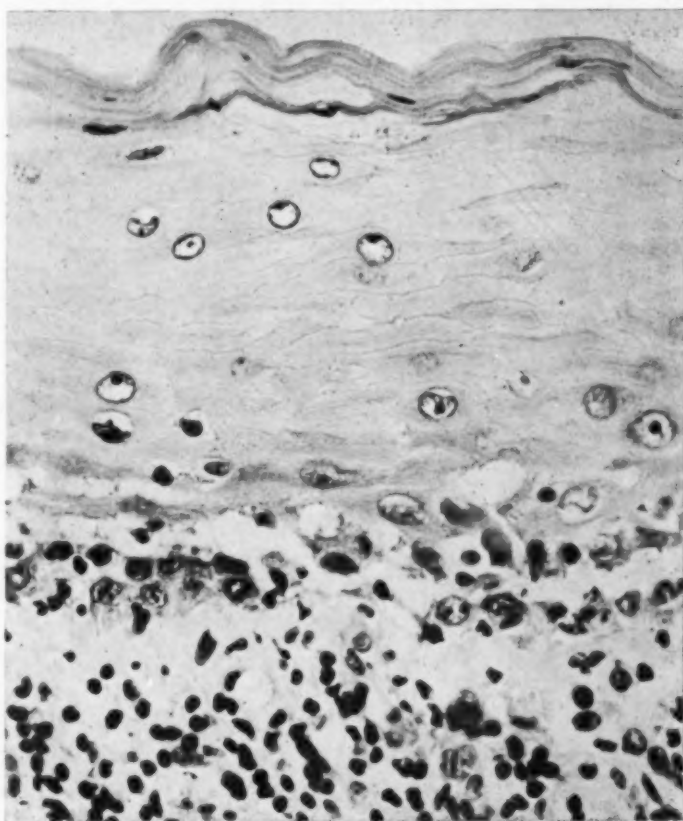


Fig. 22.—Case 9. Same tissue as Fig. 21. Upper left No. 1 interdental papilla. Areolar gingiva. Before treatment. Shows a layer of keratin on surface; compression of epithelium; hydropic degeneration of nuclei; moth-eaten appearance of basal cell layer; disappearance of the epithelial pegs; marked edema and inflammatory reaction.  $\times 640$ .

One month later, after approximately 8,000,000 vitamin A units had been administered, the lichen planus lesions disappeared. Intramuscular vitamin A therapy was discontinued and oral vitamin A was reduced to one capsule of 25,000 I.U. three times weekly. Within three weeks, subjective and objective symptoms reappeared. The dosage of vitamin A was increased to 25,000 units daily and after three weeks, symptoms again disappeared. Treatment was maintained at this dosage. Self-application of the estrogen ointment was continued throughout the treatment period.

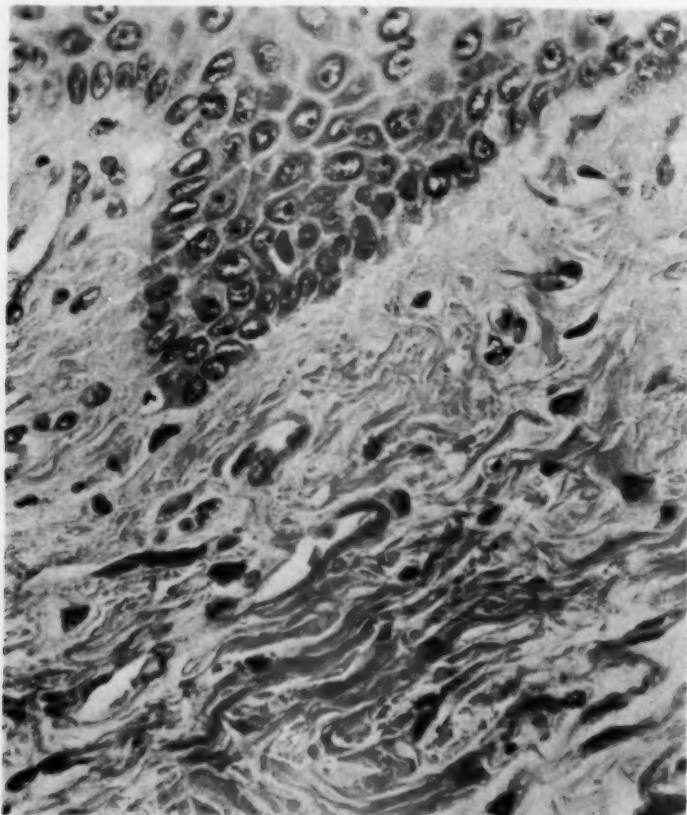


Fig. 23.—Case 9. Same case as Fig. 22 about one year after treatment with estrogen was started. Biopsy was taken from same area as previously (upper left No. 1 interdental papilla). Areolar gingiva in comparable location to Fig. 22. Shows regeneration of epithelial pegs, normal-appearing basal cells and intercellular bridges. The epithelium appears normal. There is almost complete absence of inflammatory cells and edema. The corium appears more nearly normal.  $\times 650$ .

**Results.**—With the topical applications of the estrogenic hormone, small patches of epithelization of the desquamated areas were noted after the first few weeks. These resembled small skin grafts which later coalesced to form a continuous membrane. At the outset, this membrane was thin, permitting the still intense red color of the former desquamation to remain visible. Gradually the redness dimmed. After four months, the lesions were improved to the point where they were scarcely visible in most places (see photomicrographs, Figs. 21, 22, and 23). The stiffness and soreness disappeared. The patient was allowed to resume toothbrushing.

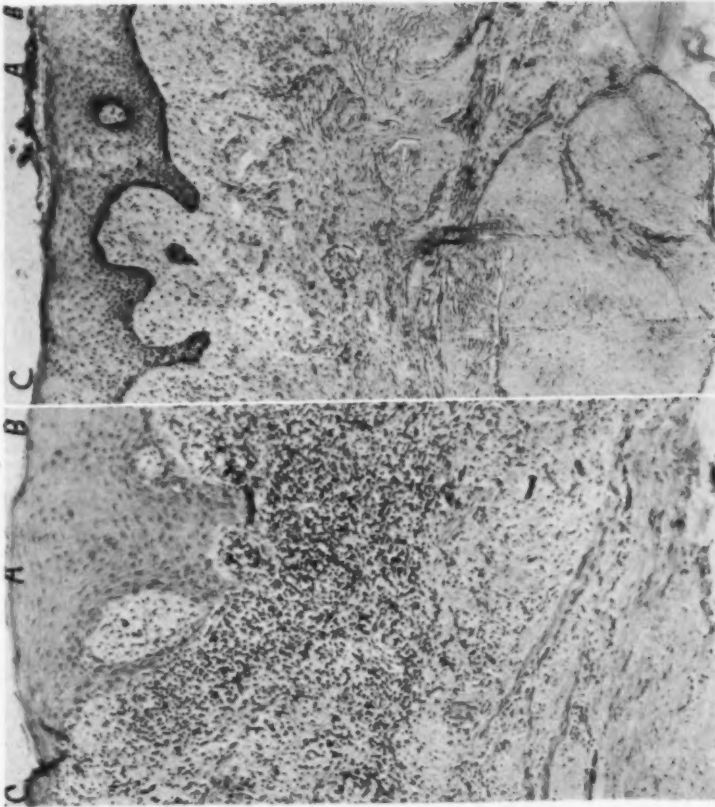


Fig. 24.

Fig. 24.—Case 9. Lower left No. 3 interdental papilla. Pretreatment, showing alveolar and areolar gingiva. A to B, alveolar. A to C, areolar. Note keratin in both areas, also intense inflammatory reaction in corium.  $\times 94$ .

Fig. 25.—Case 9. Same as in Fig. 24. Lower left No. 3 interdental papilla, after two years of treatment showing disappearance of keratin and healing of corium.  $\times 86$ .

Fig. 26.—Case 9. Higher power of Fig. 24. Areolar gingiva (pretreatment). Note particularly abnormal presence of keratin on surface.  $\times 490$ .

Fig. 27.—Case 9. Higher power of Fig. 25. Areolar gingiva, after two years of treatment. Note disappearance of keratin on healing and reappearance of a healthy basal cell layer with clear intercellular bridges.  $\times 386$ .

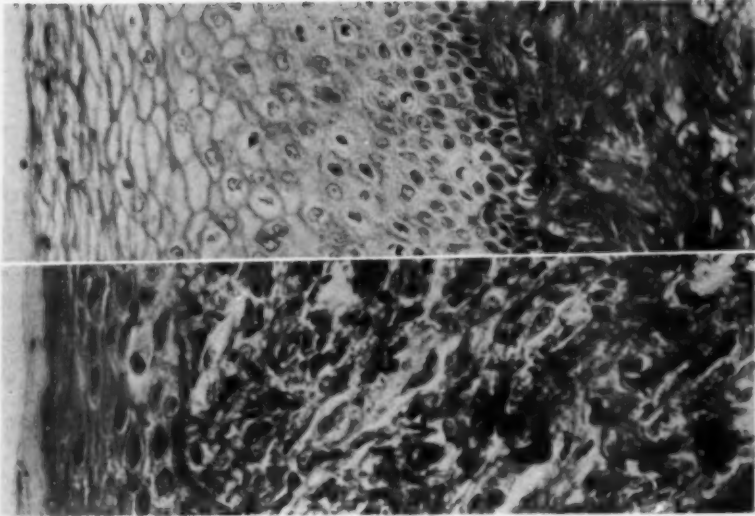


Fig. 26.

Fig. 27.

Fig. 27.—Case 9. Higher power of Fig. 25. Areolar gingiva, after two years of treatment. Note disappearance of keratin on healing and reappearance of a healthy basal cell layer with clear intercellular bridges.  $\times 386$ .

While there was some slight benefit to the lichen planus lesions from the estrogenic therapy alone, they did not disappear entirely until about five months after vitamin A was added. When the vitamin A was reduced, and while the estrogens were maintained at the same level, both the subjective and objective symptoms reappeared. By increasing only the vitamin A the lichen planus lesions again disappeared.

The desquamated lesions improved progressively throughout the entire treatment period. At this writing, the patient has been free of all oral lesions for over a year. Her present regime consists of 25,000 units of vitamin A by mouth daily and daily home applications of estrogen (see photomicrographs, Figs. 24, 25, 26, and 27).\*

CASE 10.—E. W., a Negro woman, married, aged 67 years.

*Complaint.*—Patient applied for complete mouth rehabilitation. At that time (four and one-half years ago) certain lesions were noted, suggestive of desquamative gingivitis.

*Medical History.*—Except for occasional constipation, arthritic-like pains in the back of the head, and onset of menopause at the age of 52 years, the medical history was essentially negative.

*Dental History.*—Dental care had been infrequent.

*Dental Examination.*—All lower molars and upper first molars were missing. There were several cavities and fillings. Shallow periodontal pockets were seen, especially in the regions of the upper right and left lateral incisors. Gingival recession was apparent about several teeth. There was some gingival swelling with inflammation but little bleeding. The gingivae were said to be tender to solid foods.

On the buccal surface of the gum over the upper right cuspid area, a vesicle about 1 cm. in diameter, filled with a clear serous fluid, was seen (Figs. 28 and 29). The remnants of a similar vesicle were present over the upper left cuspid area, and another small bleb with hemorrhagic fluid was noted in the alveolar gingiva opposite the lower right incisor. Patient believed these occurred after traumatization. The epithelial covering of these vesicles was easily broken, leaving a shallow ulcer.

*Treatment.*—Administration of 20 mg. of ascorbic acid, daily (supplemented by orange and tomato juices), increased to 100 mg. daily one week later and continued for three weeks, brought no improvement. In all, approximately 2,500 mg. of ascorbic acid were ingested. At this time epithelial stripping was easily provoked and new blebs formed on manipulation of the tissues. No change was noted after periodontal treatments.

Treatment with estrogen ointment for three months resulted in the disappearance of the bleb formations. The ointment was discontinued. During the succeeding four months no new blebs were seen. Subsequently, new vesicles of a similar character recurred. Vitamin B complex tablets were prescribed for three weeks. There was no apparent change. A second series of estrogen ointment treatments was given consisting of 49 applications over a period of four months. Vesicles and ulcerations disappeared. No new blebs appeared. Therapy was discontinued. The patient was examined four months later. No vesicles were

\*This case is reported in greater detail in the *Journal of Periodontology*, in press.



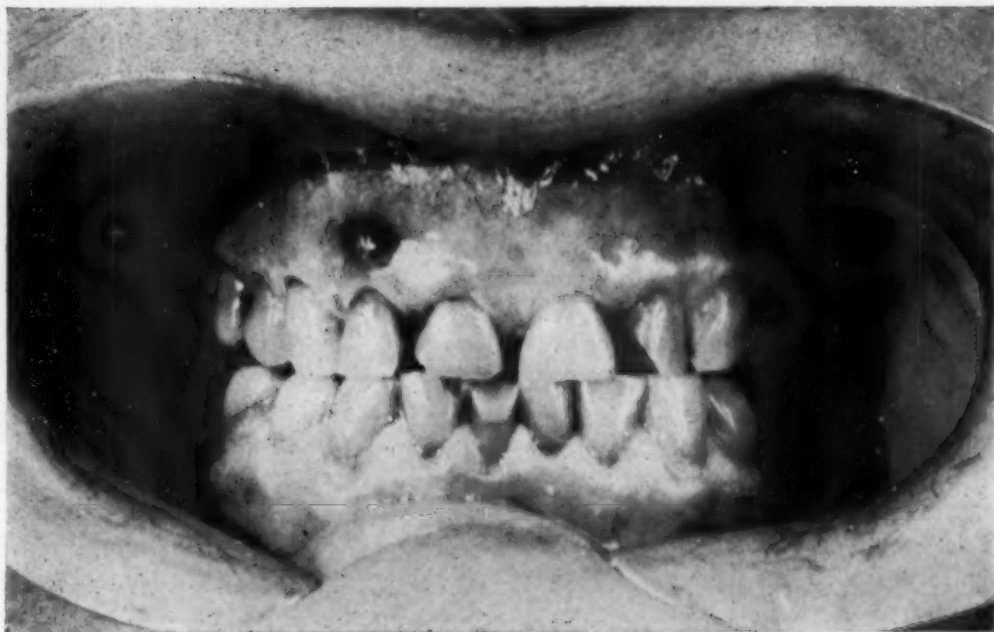


Fig. 28.—Case 10. Type 1 desquamative gingivitis. A vesicle is present on the gingiva mesial to the upper right cuspid.

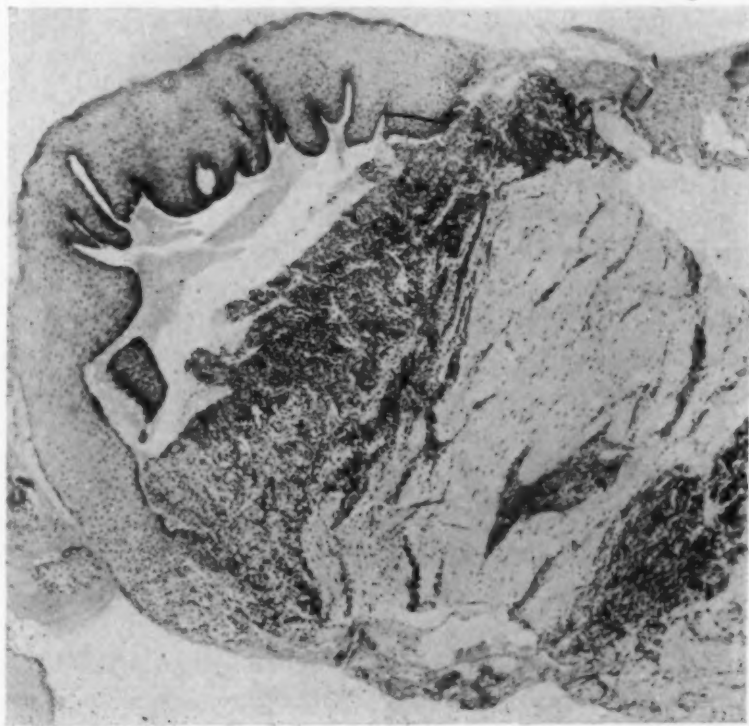


Fig. 29.—Case 10. Microscopic pathology of the vesicle shown in Fig. 28. Type 1 desquamative gingivitis forms upon rupture of a vesicle. Note the accumulation of fluid and inflammatory exudate under the epithelium causing its separation from the connective tissue and flattening of the epithelial pegs.  $\times 75$ . (Compare with Fig. 2.)

present. The patient stated that at the end of the treatment she was able to massage her gums regularly without causing injury to the surface epithelium.

*Diagnosis.*—Chronic desquamative gingivitis of the vesicular variety (Type 2).

*Follow-up Note.*—Patient returned after an absence of two years, during which time she received no treatment. The gingivae, which were normal to this time, again were forming hemorrhagic vesicles when irritated by trauma. Administration of estrogen ointment was resumed, and after three months of home application the vesicles stopped forming. Patient is still under treatment.\*

CASE 11.—H. W., a white woman, single, aged 25 years.

*Complaint.*—Pain and bleeding of gums on brushing of the teeth. Duration, about six months.

*Medical History.*—Patient suffered from convulsions at intervals for eight months after birth; mastoidectomy at the age of 5 years; retropharyngeal abscess at 7 years of age; appendectomy at 15 years of age. One year ago, the patient had profuse hemorrhage from bowel with bloating and constipation. No hemorrhoids were seen. This episode covered six to eight months. Patient also stated she was anemic at that time. For years she complained of a nervous cough associated with precordial suffocation accompanied by palpitation. There was a long-standing edema of the ankles, especially in the summertime. Patient stated she was always nervous, sometimes remaining awake all night. For the past six years she has been taking thyroid medication. During this period she tired easily, and gained weight. Menses were irregular.

*Dental History.*—Anti-Vincent's treatment for six months prior to our examination.

*Medical Examination.*—Pupils, throat, heart, lungs, abdomen, pelvic, and rectal examinations were normal or negative. Results of laboratory tests were: Kline test, negative; blood chemistry, normal; sedimentation rate, normal; blood count, normal; urinalysis, negative; guaiac stool test, negative. The basal metabolic rate was found to be -25 per cent. The fasting serum cholesterol was 216 mg. per cent. Patient was taken off thyroid medication at this time. Three months later, the basal metabolism was -22 per cent and cholesterol 229 mg. per cent.

*Dental Examination.*—The mouth was clean. All the teeth were present except the lower right second premolar and the third molars. The upper right central incisor and first molar, upper left central incisor and first molar, lower right central and lateral incisors and first molar, and lower left central and lateral incisors had hypoplastic enamel in the incisal or occlusal thirds. Several metallic fillings were present. There was a marked overjet of the anterior teeth.

The alveolar gingivae were generally of good color and texture. For the most part the marginal gingivae were inflamed and slightly swollen. The interproximal papillae were engorged and inflamed, with pseudopapillae present between the upper right central and lateral incisors, upper right and left central incisors, upper left central and lateral incisors, and upper left lateral incisor and cuspid.

\*This case was treated by Dr. F. Beube of Columbia University through whose courtesy we are presenting it here.

Red, raw, beefy areas of desquamation were seen in the mucobuccal fold opposite the lower right and left second premolars, and in the upper jaw opposite the right second premolar and left first molar.

*Diagnosis.*—Chronic desquamative gingivitis of the atrophic variety (Type 2).

*Treatment.*—Patient was placed on estrogenic ointment medication once a day for two hours over a period of almost three months. At the end of this time the desquamated areas had disappeared entirely. Early in the treatment period the physician in the case prescribed vitamin B complex. Soon thereafter, necrotic sloughs, characteristic of acute Vincent's infection, were seen on the gingivae. Vitamin B therapy was discontinued and the Vincent's symptoms disappeared.

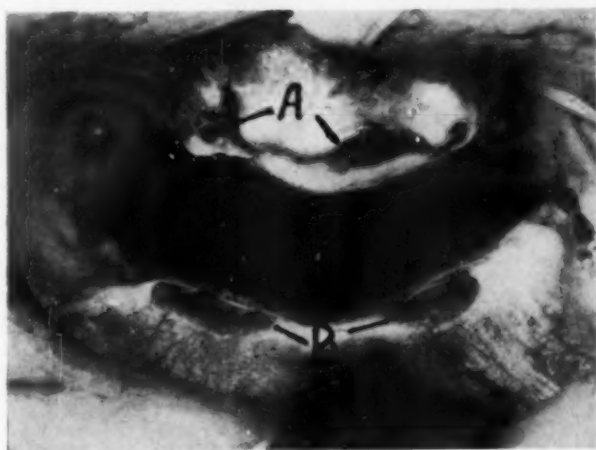


Fig. 30.—Case 12. Type 2 desquamative gingivitis is present on A the upper, and B the lower edentulous ridges. The lesion developed following the extraction of the teeth.

CASE 12.—C. S., a white woman, married, aged 43 years.

*Complaint.*—Pain and a sensation of enlargement of the gums following full mouth extraction.

*Medical History.*—Patient had given birth to ten children. She had had an appendectomy, and stated that she went through menopause three years previously. Otherwise, the medical history was negative.

*Dental History.*—All teeth were extracted two and one-half months prior to our examination. Patient took one multivitamin capsule daily for two months. There was no improvement of the gingival lesions.

*Dental Examination.*—Areas of desquamation about 1 cm. in width were noted along the crests of the upper and lower edentulous posterior ridges and at intervals along the crests of the anterior ridges (Fig. 30).

*Diagnosis.*—Chronic desquamative gingivitis of the atrophic variety (Type 2) (Figs. 31 and 32).

*Treatment.*—Estrogen ointment was applied daily in the manner described. After two months the gingival lesions had disappeared completely in all but three small areas in the regions of the upper right premolar, lower right pre-

molars, and the lower left incisor. In these sections the gums showed definite improvement. There were signs of epithelization occurring in patches and tending to cover the surfaces of the formerly desquamated areas.

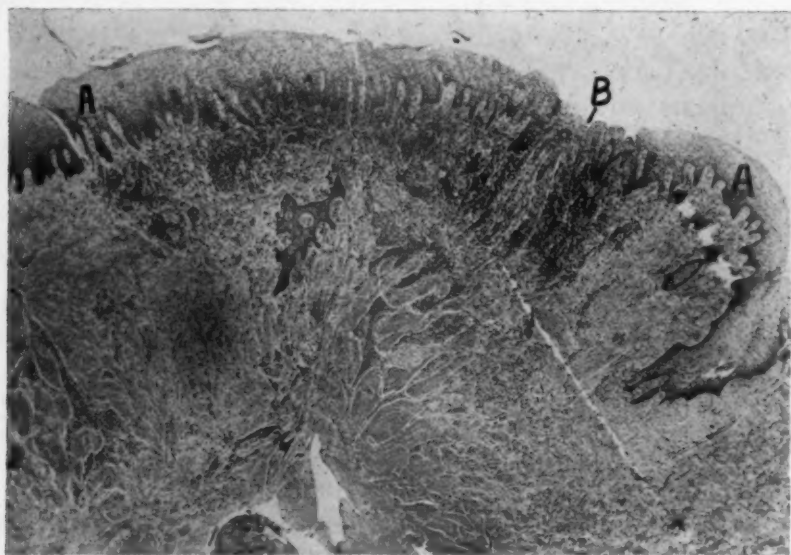


Fig. 31.—Case 12. Microscopic pathology of the desquamated areas shown in Fig. 30. Normal areas are seen at A and epithelial breakdown at B.  $\times 37$ .

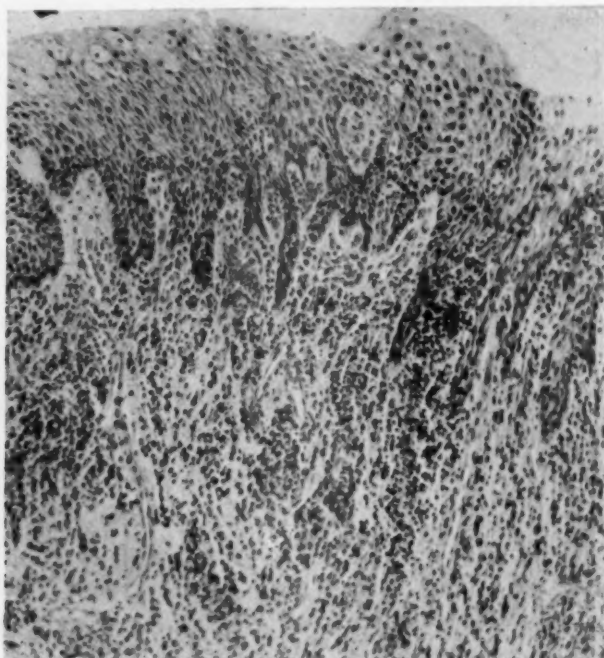


Fig. 32.—Case 12. Higher power of Fig. 31. Note marked hydropic degeneration of epithelium.  $\times 150$ .

#### MICROSCOPIC PATHOLOGY

*Epithelium.*—In most of the cases studied, the areas of involvement were wholly or partially denuded of epithelium (Figs. 2 and 8). Some retained thin



strips or patches of epithelium in active stages of degeneration (Figs. 7 and 26). Generally, the keratin layer was absent. Parakeratosis was seen at intervals (Fig. 6). Hydropic degeneration was prominent throughout the epithelium remaining over the involved areas (Figs. 4, 22, and 32). The intracellular edema was accompanied by intercellular edema, the latter causing disintegration of the intercellular bridges (Figs. 4 and 6). There was marked edema in the corium contiguous to atrophic epithelium with subsequent necrosis and loss of epithelial covering of the corium. In some regions the "pavementing" characteristics were still discernible (Fig. 26). In Cases 1, 3, 9, and 11 (Figs. 9, 22, and 26) the presence of a definite, well-defined layer of keratin was observed on the areolar gingivae and the buccal mucosa. Under this layer was a stratum granulosum (Fig. 22). (Keratin is not ordinarily seen in these regions.) These findings were present beyond the limits of the desquamated areas with no clinical evidence of hyperkeratosis. In Cases 2, 5, 8, and 12, these signs of metaplasia were absent. In the remaining four cases, 3, 4, 7, and 10, specimens of the area were not available.

*Corium.*—In the cases of long duration the corium showed large areas denuded of epithelium. In those of short duration the papillae were extremely close to the outer surface (Figs. 4, 31, and 32). The apices of the papillae were composed mainly of an edematous fibrillar connective tissue containing many dilated blood vessels (Fig. 4). Widespread inflammation was observed throughout the corium in areas seemingly uninvolved clinically (Figs. 31 and 32). Fragmentation of the collagenous connective tissue bundles was common, as were dense masses of inflammatory cells which were composed of round cells and plasma cells (Figs. 16 and 24). In some regions the inflammation completely displaced the connective tissue (Figs. 4, 26, and 29). Vascularity of the corium was markedly increased (Fig. 32). Areas of laked blood were seen in the outer layer of the dermis.

*Variations.*—Two definite forms of desquamation were discernible in these studies, both grossly and microscopically.

In the first type, referred to here as the "vesicular," an accumulation of fluid was observed in the subepithelial layer (Fig. 29). The fluid was localized and showed a gradual increase in amount and in internal pressure. Eventually, the enlargement expanded outwardly causing the epithelial pegs to become blunt and flat and the entire overlying epithelial layer to stretch thinly across the raised lesion. The vesicle varied in size from a few millimeters to 4 or 5 cm. in diameter. There were evidences of pressure atrophy and rupture of the outer epithelial covering of the vesicle (Fig. 2).

The second type of desquamation, "atrophic," was characterized by a stripping or sloughing off of epithelium in sheets. There was widespread, diffuse edema in the epithelium and underneath it (Figs. 9, 21, and 22). An intense chronic inflammatory process was seen in the corium. Both the edema and the inflammatory exudate were contiguous to atrophic changes in the basal cell layer, accompanied by flattening of the epithelial pegs. The degenerated epithelium was fragile, as evidenced by the fact that the tissues tore easily in handling (Figs. 4 and 13).

In Type 1, the localized fluid produces the vesicle. In Type 2, the edema is diffused over a more widespread area. In other respects the two are similar.

#### DISCUSSION

As a result of histological study of the various areas of the affected gingivae, an attempt was made to reconstruct the progress of the disease to its final destruction of the tissue.

The probable field of origin is the papillary layer of the corium, where the greatest amount of involvement was seen. Deeper in the corium some parts were affected while others were not. Microscopically, the most marked inflammatory reaction occurred under the clinical lesion with less intense tissue changes beyond its limits. The mildly affected areas, unobscured by severe edema and necrosis, and appearing normal grossly, showed evidence of the early stages of the disease. The initial signs were increased prominence of the capillary bed, scattered foci of inflammation, and small (microscopic) sub-epithelial vesicles. The edema and the inflammation became progressively worse as closer proximity to the clinical lesion was reached. The edema produced a pressure atrophy of the basal epithelial cells and the inflammatory reaction invaded the epithelium. Intercellular and intracellular edema caused further destruction of the epithelium and rendered the tissues more friable. Thus, in some instances, the surface covering was lost through stripping, owing to the weakened condition of the epithelium or to extension of the inflammatory process and atrophy.

Intraepithelial vesicles were also thought to cause loss of large areas of epithelium. Lesions of the character under discussion are found in areas where trauma is common, not only in the oral cavity but in other parts of the body. Pertinent to this interpretation are observations made by Gross<sup>10</sup> who reported similar lesions in diseases where trauma, friction, or stretching might be eliciting factors (hairline, nasolabial folds, under breasts, vagina, anus, elbows, knees, groin, scrotum, vulva, etc.).

The factors initiating these changes are seemingly of systemic origin even though the patient's medical history may be negative. The local disturbances of the mucous membranes and skin, oral and in other parts of the body, may be the only clinical manifestations of a functional imbalance. The systemic implications are based on significant data.

The presence of scattered foci of inflammation in the corium of normal-appearing mucous membranes, in localized areas, points to a disturbed metabolism. The presence of a definite well-developed keratin layer on the areolar gingivae and buccal mucous membranes, not found normally, may be another indication of metabolic disorder. In a previous report<sup>11</sup> this condition was found to be prevalent in hypothyroid human beings and monkeys. Stripping of the epithelium was also seen in sections of gingivae from thyroidectomized monkeys.<sup>12</sup> In the present study hypothyroidism was noted in some cases. While not constant, it appeared often enough to suggest a possible etiological relationship. Clinically, it might account for the skin changes seen in three cases, the overweight findings in six, the lowered basal metabolism in two, and the mental retardation in one. It might also have influenced the irregular menses found in

three cases. These ideas support the hypothesis that desquamative gingivitis may be accompanied by a disturbance of some metabolic process.

The fact that the gingivae are benefitted by the application of the female sex hormone suggests a deficiency of the hormone in this area as an etiological factor. Irregular menses in three cases and the frequency of observed exacerbations of the lesions during the menstruation period, at a time corresponding to low estrogen tide, lend emphasis to this interpretation. Even the presence of normal menses or the occurrence of desquamative gingivitis in men does not negate the theory. By comparing oral and vaginal epithelial smears, it has been found that the estrogen threshold of the oral mucous membranes is much higher than that of the vagina.<sup>13</sup> That is, under estrogen therapy the vagina reacts before and to a greater degree than do the oral mucous membranes. Other investigators have reported variations in the estrogen threshold of the tissues in different parts of the body.<sup>14, 15</sup> The implication is that there may be enough estrogen biologically active to maintain normal menstruation but not enough to prevent oral desquamation.

Estrogen as a factor is suggested in still another way. In Cases 1 and 11, there were exacerbations of the mouth symptoms coincident with the taking of vitamin B complex. Since then, we have observed in other cases the sudden appearance of acute gingival inflammation with marginal necrosis following the prescription of vitamin B after a discontinuance of thyroid medication.

In Case 1 the desquamated lesions were under control for a number of years. A relapse was coincident with the self-administration of vitamin B complex. Following the discontinuance of the vitamin B, the pain symptoms and ulcerations gradually subsided without medication. When vitamin B was prescribed for this patient a year later, in the treatment of a systemic condition, the acute oral symptoms returned. They were relieved by application of estrogen ointment. The explanation for this would seem to lie in the reported findings that vitamin B depletes endogenous estrogen by inactivating it in the liver, thus producing estrogen depletion in the mucous membranes. Biskind and Mark<sup>16</sup> and Biskind and Shelesnyak<sup>17</sup> demonstrated that the estrogen inactivating mechanism of the liver may be impaired at a stage of vitamin B complex deficiency when the ovary is still functional. They stated that the liver may have an important part in maintaining the normal level of estrogen in the body.

By implanting pellets of estrone in the spleens of adult castrate female rats, Biskind and Biskind<sup>18</sup> showed that animals on a normal diet remained anestrus. This would indicate that the estrogen in the implanted pellets passed through the liver before reaching the circulation, and was there inactivated. When this experiment was repeated with rats on a diet free of vitamin B complex, protracted estrus ensued. Addition of brewer's yeast to the diet again caused the animals to become anestrus. Subsequent dietary vitamin B depletion led to estrus.

Ashworth and Sutton<sup>19</sup> reported that the administration of estrogens to patients with subclinical vitamin B complex deficiencies may cause the appearance of lesions such as polyneuritis, pellagra, cheilosis, etc. This was demonstrated in alcoholics with cirrhotic livers who showed marked symptoms of vitamin B deficiency. When given vitamin B for varying periods, the symptoms improved. After substituting estrogen for the vitamin there were severe recur-



rences of the deficiency symptoms. Discontinuing the estrogens and returning to the B complex brought alleviation. These authors also reported that symptoms of pellagra tended to increase during the menstrual cycle. Zondek<sup>20</sup> showed the oxidation of estrogen by *in vitro* experiments. Estrogen was quickly destroyed when placed in cultures of fresh infantile rat-liver tissue.

These experiments show an interrelationship between the estrogens and vitamin B complex in their metabolism in the liver. The study reported here concludes that relative vitamin B excesses producing endogenous estrogen depletion may react adversely on the oral mucous membranes.<sup>21, 22</sup>

The use of estrogen in the clinical management of desquamative gingivitis was an outgrowth of previously reported studies<sup>23</sup> on the effect of this hormone on oral mucous membranes. With its parenteral use in both monkeys (experimentally) and human beings (in treatment of gynecologic disorders), hyperkeratinization and hyperplasia of both epithelium and connective tissue were absorbed. These results were regarded as beneficial because of the improved appearance of the tissues clinically and the reduction in inflammatory exudate microscopically. Estrogen ointments applied topically were found to be even more efficient. We first reported their use in this way in 1939.<sup>23</sup> Mortimer, Wright, and Collip<sup>24</sup> in 1937 advocated the use of the insufflation method of applying estrogen in cases of atrophic rhinitis, and Buxton<sup>25</sup> in 1942 advocated the use of estrogen ointment in the treatment of kraurosis vulva and other vulval disorders.

The possibility of estrogen producing a localized reparative metaplasia similar to the changes reported by Bessey and Wolbach<sup>26</sup> in instances of vitamin A deficiency might be examined. These alterations are described as "Atrophy of the epithelium concerned, reparative proliferation of basal cells, and differentiation of the new product into a stratified keratinizing epithelium. This replacement epithelium, regardless of the previous formation and structure in the region, is identical in all locations and comparable in all its layers with epidermis." It will be seen that the "estrogen" mucous membrane changes and the vitamin A deficient mucous membrane changes are analogous.<sup>27</sup> Conversely, estrogen has a physiologic action in its normal production of hyperplasia and hyperkeratinization of vaginal mucous membranes during the menstrual cycle and estrus. Estrogen also restores to normal atrophic vaginal mucous membranes in women suffering from estrogen deficiency states (as in menopause) or ovarian dysfunction. Therefore, credence may be given the hypothesis that the estrogen treatment as described here restores an essential element to the oral tissues.

The constant need of estrogen for the proper maintenance of the health of the oral mucosa may explain the relapses when therapy was discontinued or its biologic action nullified in other ways.

#### SUMMARY

Twelve patients, aged 21 to 67 years, of whom ten were women and two men, presented with symptoms of desquamative gingivitis. A comprehensive review of the literature on this disease is included. Case reports illustrated with photographs and photomicrographs are given. The clinical and histopathologic findings are described. A method of clinical management and its rationale are discussed.



The disease is hypothetically designated as a local manifestation of a metabolic disturbance. Various causes of the disturbed metabolism are considered. These include especially the abnormal functioning of the thyroid gland and the interrelation of the vitamins and estrogens. The data suggest that a local depletion of estrogen in the oral tissues may play a major causative role. Estrogen ointments applied topically are effective in controlling the disease.

We wish to express our thanks to Dr. Schwenk and Dr. Gilbert of the Schering Corporation for their many courtesies, and to Dr. H. Silvers who assisted in the treatment of some of these cases.

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## THE CHOICE OF TREATMENT FOR PERIODONTAL POCKETS

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MANY approaches and methods have been devised for the treatment of periodontal disease. Fundamentally, all schools agree on the necessity of restoration of function, institution of proper nutrition, and attention to systemic factors, as well as the removal of gingival irritants and other etiological factors. The only point of difference in rationale is in the approach to the elimination of the pocket itself. Hundreds of methods and therapeutic agents have been devised to accomplish this procedure. Most were expounded vehemently only to be dropped after more careful trial.

Essentially, there are three broad categories into which all procedures of pocket elimination fall:

1. Radical elimination.
2. Conservative surgical procedures.
3. Conservative subgingival curettage.

In the handling of any periodontic case the first procedure should always be conservative. It is surprising to see how pockets shallow out and gingival tone is restored after balancing of the occlusion, subgingival curettage, and intelligent toothbrushing procedure. With such simple combined therapy, a marked return to health may be expected. In most cases the return to health is complete enough to make more radical procedures of pocket obliteration unnecessary. Anyone who can be trained to carry on the usual operations of dentistry can attain the ability to perform the three afore-mentioned essential phases of treatment. For that reason the general practitioner should be able to handle periodontal cases just as successfully as he does other branches of his calling. When it is realized that more than half of the teeth extracted are lost due to periodontal disease, the opportunity for increased service is apparent.

There are, however, some cases which present at least one or more pockets which do not respond to the conservative procedures of balancing, instrumentation, and toothbrushing, or react so slowly to these means that more radical steps are necessary. Also, it can be conceived readily that some pockets may occur which are not accessible for conservative subgingival curettage, or that the skill required to treat them would not be available to the average practitioner. It is therefore no more justifiable for a periodontist or general practitioner to be purely a conservative operator than for a physician to consider that surgery would never be needed in his cases.

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## RADICAL POCKET ELIMINATION

Four general groups fall into this category: (1) surgical; (2) electrical; (3) chemical; (4) mechanical.

1. *Surgical: Ward Technique.*—Elimination of pockets down to solid bone, creating a new continuous gingival margin free of pockets and decalcified bone.

*Crane-Kaplan Technique.*—Elimination of pockets as in the Ward technique, the line of excision following the festooning of the pockets rather than producing a straight gingival line immediately subsequent to operation.

*New Kirkland Operation.*—Essentially the same as the Crane-Kaplan operation but following the pocket outline on the facial and an arbitrary 3-mm. reduction on the lingual.

*Black Operation.*—Reduction to the gingival attachment, not actually involving bone or perforating the fundus of the pocket.

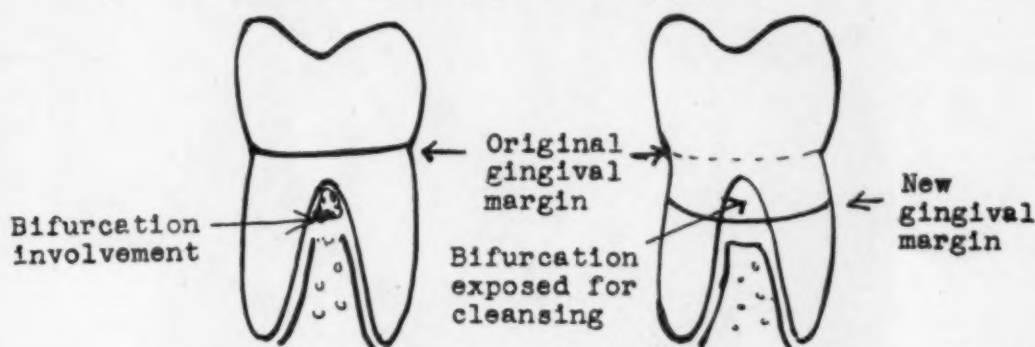


Fig. 1.—Method of treating a lower first molar with involvement at bifurcation by reducing its gingival margin below the involved area, thus creating two individual roots resembling bridge abutments, the crown acting as a bridge with the bifurcation exposed for cleansing.

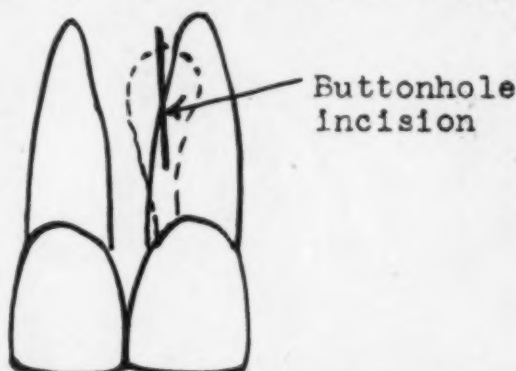


Fig. 2.—Buttonhole incision. This is successful for the treatment of pockets, the fundus of which cannot be reached readily through the marginal orifice. To avoid recession the incision must not extend closer to the gingival margin than 3 millimeters. The superficial part of the pocket is curetted through the natural opening, the deeper less accessible part through the incision.

2. *Electrical.*—The pocket wall may be trimmed by the monopolar cutting current or by the electrocoagulation bipolar technique of Webb.

3. *Chemical.*—Any caustic chemical can be used to reduce pockets by direct application. Unfortunately many of these, e.g., nitric acid, trichloroacetic acid, and chromic acid, are injurious to tooth structure.

4. *Mechanical.*—Atrophy of the soft-tissue wall of the pocket can be accomplished by the compression of the gingival tissue by paraffin wax as ad-

vocated by Dunlop. Baseplate gutta-percha, zinc oxide-eugenol cements thickly mixed or combined with absorbent cotton, and similar materials may be used as the compressing agents.

#### INDICATIONS FOR RADICAL POCKET ELIMINATION

1. For pockets which do not heal sufficiently after conservative treatment either by reattachment or marginal atrophy so that the resultant sulcus is less than 1 mm. in depth.

A.



B.

Fig. 3.—Effect of conservative treatment in a man, aged 38 years. A, Before treatment; B, after treatment.

Note change in gingival contour as well as improvement in tooth form and appearance accomplished by reshaping. The overbite was excessive and complete balance was not attainable in this case. The change in angle of incidence of the biting forces and the reduction in leverage aided healing.

2. To create exposure of insufficiently erupted teeth either to make them more readily cleansable or to increase the length of the exposed crowns so that inlays, clasps, or other abutment attachments can be fitted properly.

3. For lower molars with bifurcation involvements in which the roots are widely spread and the tissue can be so reduced as to permanently expose the bifurcation for cleansing (Fig. 1).



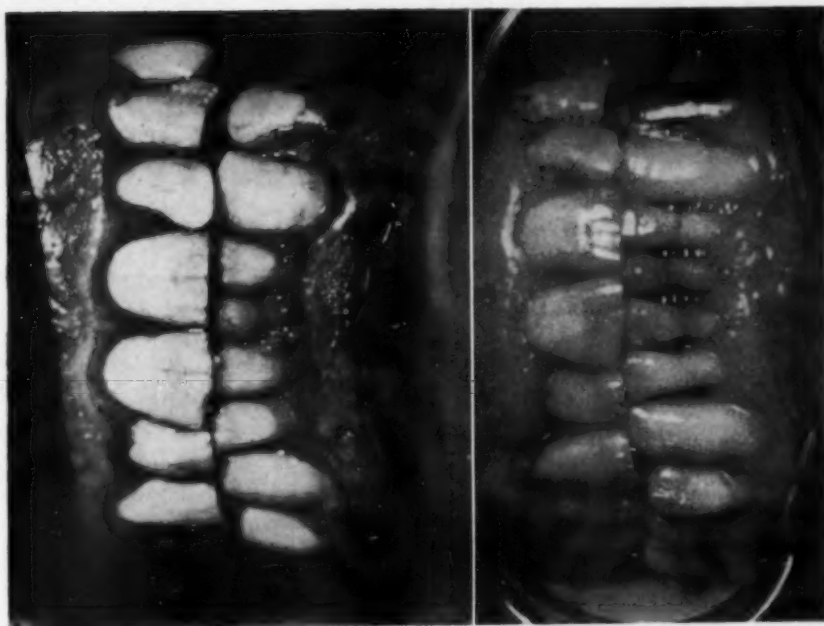


Fig. 5.

Fig. 5.—Change in gingival form resembling surgical gingivectomy on lower anteriors after conservative treatment. If the cause is eliminated and the correct procedures employed, tissues that cannot reattach will resorb to a safe equilibrium with the underlying bone. *A*, Before treatment; *B*, after treatment. (Courtesy W. M. Greenhut.)

Fig. 6.—Man, aged 64 years. Upper teeth were treated conservatively; lowers by gingivectomy. Note that there is much less recession in the upper arch, creating a safer, more protective gingival contour. *A*, Before treatment; *B*, after treatment. (Courtesy S. Sorrin.)

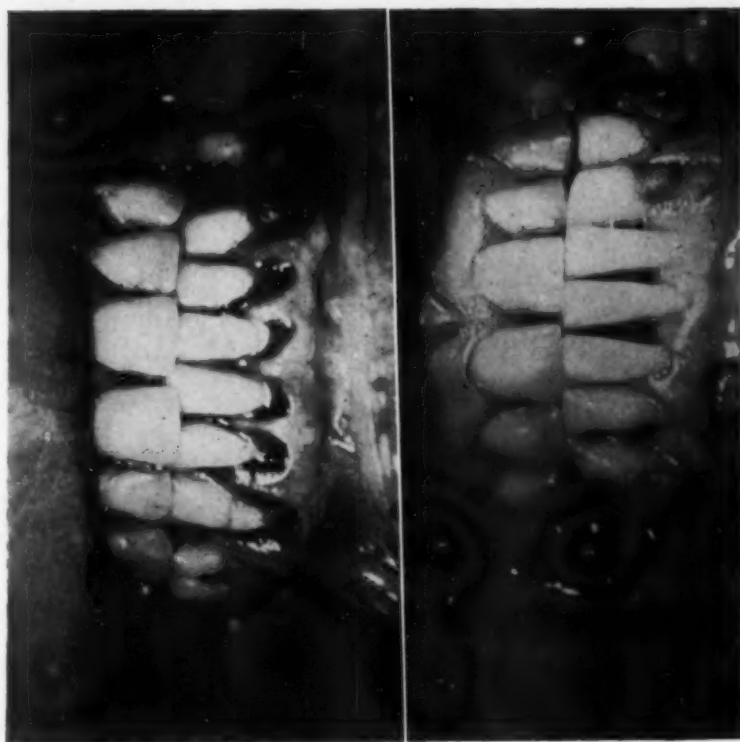


Fig. 4.

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*A*.

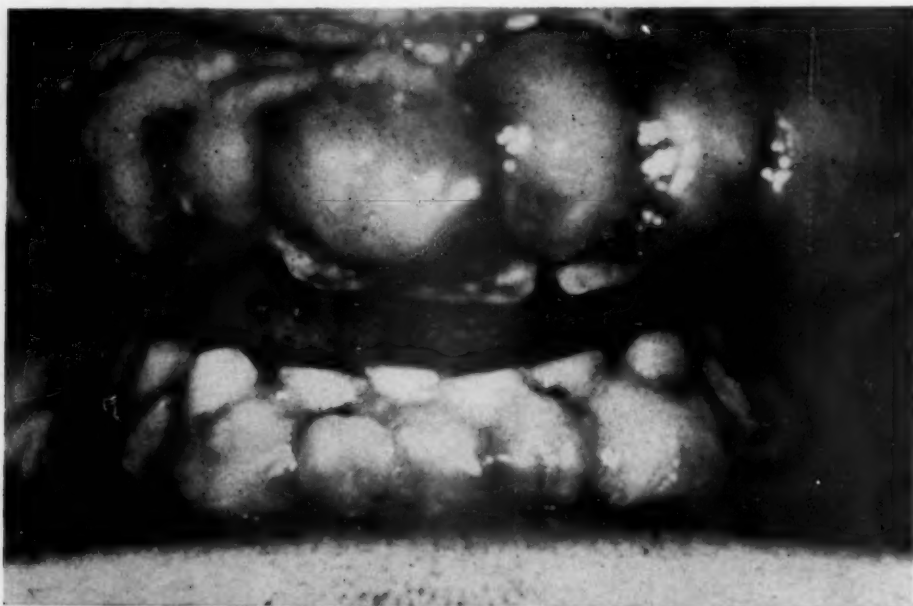
*B*.

4. In eliminating a flap over incompletely erupted third molars when the teeth are in proper alignment and the crowns are not kept from complete eruption by bone on the distal.

#### CONSERVATIVE SURGERY

1. *Flap Operation (Rubicek).*—Vertical incisions are made on each side of the pocket to be operated on, the gingival margin is freed, and the tissue is retracted so as to expose the involved area.

A.



B.

Fig. 6.—Dilantin hypertrophy. A, Before conservative treatment; B, after conservative treatment.

Therapy consisted of curettage and toothbrush stimulation with some balancing. No surgery was employed. Dilantin was *not* discontinued. Previous surgical treatment was a failure.

2. *Original Kirkland Operation.*—Gingival margins are freed over the pockets to be operated on and the tissue is retracted. No vertical incisions are made.

3. *Buttonhole Incision (Miller)* (Fig. 2).—A vertical incision is made over the deepest portion of the pocket *not* extending to the gingival margin. The deeper part of the pocket is curetted through the vertical incision, the superficial part through the natural pocket orifice.

INDICATIONS FOR CONSERVATIVE SURGERY

1. Tortuous pockets: in such cases it may not be possible to reach the fundus of the pocket by subgingival curettage without excessive tissue injury.
2. Pockets wider at the base than at the orifice.
3. To eliminate granulomatous tissue.

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## DYNAMICS OF WOUND HEALING FOLLOWING ELIMINATION OF GINGIVAL POCKETS

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### INTRODUCTION

THE elimination of the gingival pockets in the past has been accomplished by methods based purely on clinical observations and experience. There would be no objection to such empiricism if it had resulted in fairly uniform methods of operating. There are as many variations in methods, however, as there are operators. The literature does not reveal any comprehensive study which would determine the relative value of different procedures. The progress of all surgery has been dependent upon the addition of exact scientific knowledge to the indispensable experience gained through clinical practice.

A clinical experimental method was devised and employed to obtain fundamental information of the dynamics in wound healing following the elimination of the gingival pocket. This plan was carried out as follows: Gingivectomy was performed, under local anesthesia, in a certain area of the oral cavity. The anterior region is preferred for the investigation because of its accessibility and clear observation. Suitable patients presenting extensive involvement of the gingiva and favorable position of the teeth were selected for this investigation. The interdental spaces had to be comparatively wide so as to furnish an adequate amount of tissue for the histological investigation. In successive days, sections of the tissue were removed for the study of the healing process.

In order to explain this plan of investigation, and so enable other investigators to continue the study, a typical case is described in detail. To simplify matters, a case of gingivectomy will be used as an illustration; but gingivectomy may be substituted by electrocoagulation, chemical coagulation, or any other method which might be used for the elimination of the gingival pockets.

Gingivectomy was performed from the right mandibular first premolar to the left mandibular first premolar. The area of operation was selected according to the number of teeth present, the advance of the disease, and the accessibility for this investigation. The premolar to premolar example was merely selected to illustrate the plan of investigation.

### EXPERIMENTAL PLAN

Lower front teeth									
R	4	3	2	1		1	2	3	L
	2 days	4 days	6 days	8 days		10 days	12 days	14 days	

Two days after gingivectomy, a section of gingiva was removed from between the right first premolar and cuspid. The specimen was prepared for

Foundation for Dental Research of the Chicago College of Dental Surgery, Dental Department of Loyola University.



microscopic examination, showing the condition of the healing tissue two days after the operation.

Four days after the gingivectomy, a section of gingiva was again removed from between the cuspid and lateral incisor. This specimen showed the progress of tissue healing after a period of four days.

Six days after the gingivectomy, a specimen was removed from between the lateral and central incisors. Two days later, after eight days in all, a section was removed from between the two central incisors. Thus the final section between the cuspid and premolar on the left side was obtained after fourteen days.

The microscopic specimens secured in this manner gave evidence of the entire process of tissue healing after gingivectomy, up to fourteen days. The two-day interval was chosen only as a convenient length of time. This procedure has been followed quite frequently, shortening or lengthening the intervals of removal of sections as required for a particular experiment.

#### FINDINGS

Gingivectomy was performed in the lower anterior region, under local anesthesia. The gingival tissue was removed from around all the teeth, the amount being determined by the depth of the gingival pockets. Bleeding was stopped by the use of epinephrine gauze. No other drugs were used, and no other surgical dressing was applied. In two, four, seven, nine, twelve, and fourteen days after gingivectomy, sections of the gingiva were removed surgically and were studied microscopically.

Clinical observations revealed that healing took place rather rapidly. In the beginning, the wound surface had a grayish-colored blood clot, and later, a red, velvety granulation tissue. After fourteen days, the surface appeared to be covered by epithelium.

Histological investigation of the healing wound revealed the progress that occurred from the blood clot being cast off, to the complete epithelial covering of the wound surfaces.

The tissues with which we deal in gingivectomy are the epithelium covering the surface of the mucous membrane, and the underlying connective tissue. The connective tissue consists chiefly of connective tissue cells, fibroblasts, and fibrous intercellular substances. The fibroblasts are fixed connective tissue cells, whereas the fibers consist of collagenous, argyrophil (precollagenous), and some elastic fibers. There are numerous capillaries and small vessels in the gingiva. Besides these more or less fixed tissue elements, certain cells are present which play a very important part in the tissue reactions taking place in wound healing. These cellular elements are referred to as histiocytes (resting wandering cells), undifferentiated mesenchymal cells, and small active wandering cells (lymphocytes). The blood and its elements in wound healing must also be considered. The migrating leucocytes and red blood corpuscles, which enter the tissue during hemorrhage, and especially fibrin, which plays an important part on the wound surface and in encasing the wound area, are all essential factors in wound healing.

Fig. 1 shows the interdental gingiva between the lower central and lateral incisors in the case under discussion. It represents the microscopic picture of a

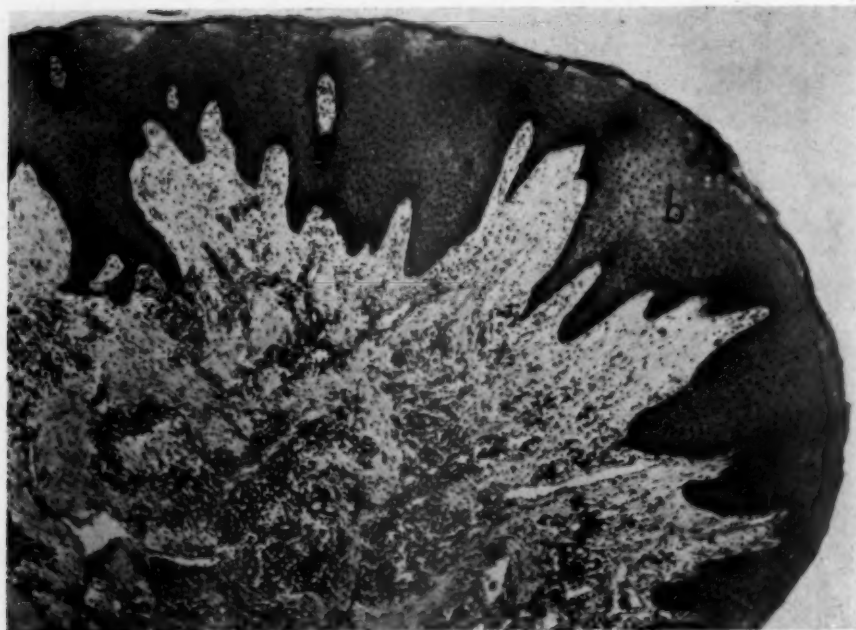


Fig. 1.—Gingiva demonstrating chronic inflammation. The connective tissue is heavily infiltrated by plasma cells (a). The epithelium (b) is thickened and the epithelial pegs are irregular (acanthosis).

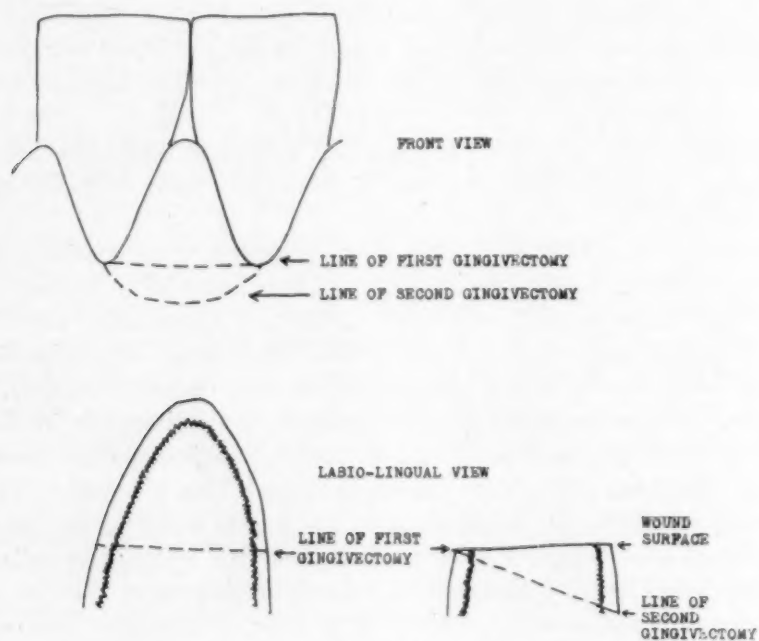


Fig. 2.—Diagram explaining the plan of investigation.

chronic gingivitis. The topography of the specimen, shown in Fig. 1, can be recognized from the diagram in Fig. 2. Fig. 1 illustrates the tissues above the line of the first gingivectomy. There is chronic inflammation in the connective tissue, borne out by the presence of plasma cells (*a*) in great abundance, with only a few leucocytes close to the surface. This accumulation of plasma cells is one of the characteristics of inflammation of the gingiva. Seldom, under any conditions, are as many plasma cells found anywhere in the body, as in the gingiva. If they are present, other than in the gingiva, it is usually a sign of specific systemic infection. The epithelial covering (*b*) of the gingiva seems to be somewhat thickened, and the epithelial pegs, penetrating into the connective tissue, are irregular in length and width. This condition in pathology is termed acanthosis. The uppermost layer of the epithelium which, under normal conditions, should be well keratinized, is poorly formed and shows signs of parakeratosis (cell nuclei within the keratin layer) with the formation of hyaline bodies (*b*). These conditions are most probably reactions to the chronic inflammation of the underlying connective tissue.

Two days after gingivectomy was performed, the interdental gingiva between the cuspid and lateral incisor was again resected according to the line, as shown in Fig. 2, for the second gingivectomy. As the diagram illustrates, the specimen obtained in this way shows the wound surface and the gingival epithelium on the labial side. By use of this method it is possible to study the condition of the epithelium at the surface of the wound, as well as the reaction taking place in the connective tissue below the wound surface.

Fig. 3 shows the healing process of the wound surface two days (*A*), four days (*B*), nine days (*C*), and fourteen days (*D*) after gingivectomy. Two days after gingivectomy (*A*) the wound surface is covered by a blood clot. On the right side of the picture, the epithelial covering of the gingiva (*a*) can be seen, and on the left side, the wound surface (*b*) covered by a blood clot (*c*). The epithelium extends into the blood clot, with a rather pointed edge, underneath which the tissue is extremely edematous (*d*), only a few cells being present. Whereas, below the blood clot, the cellular activity is very marked (*e*).

At the end of the epithelial projection the cells seem to be joined rather loosely. Fig. 4 shows a high magnification, close to the end of the epithelium. There is a mitotic figure (*a*) in the basal layer, which is in close proximity to the fibrous network of the blood clot (*b*). A large number of leucocytes have migrated toward the outer surface and have penetrated the epithelium. The round shape and loose connection of the epithelial cells, as well as the mitotic figure, indicate that there has been considerable activity in the epithelium during the two days after gingivectomy.

The blood clot has a very significant function in wound healing since it protects the exposed connective tissue from invading bacteria. Bacteria will cover the surface of the blood clot where conditions for their growth are favorable. As a result of the action of these bacteria, the surface of the blood clot becomes necrotic. The blood cells, within the fibrin network of the blood clot, lose their staining reaction and the surface layer becomes somewhat homogeneous (Fig. 3, *A*, *f*). In contrast to this outer layer, the center area (*g*) is very rich in cells, which are partly the original blood cells, and there are also a large number of leucocytes which have migrated from the underlying con-

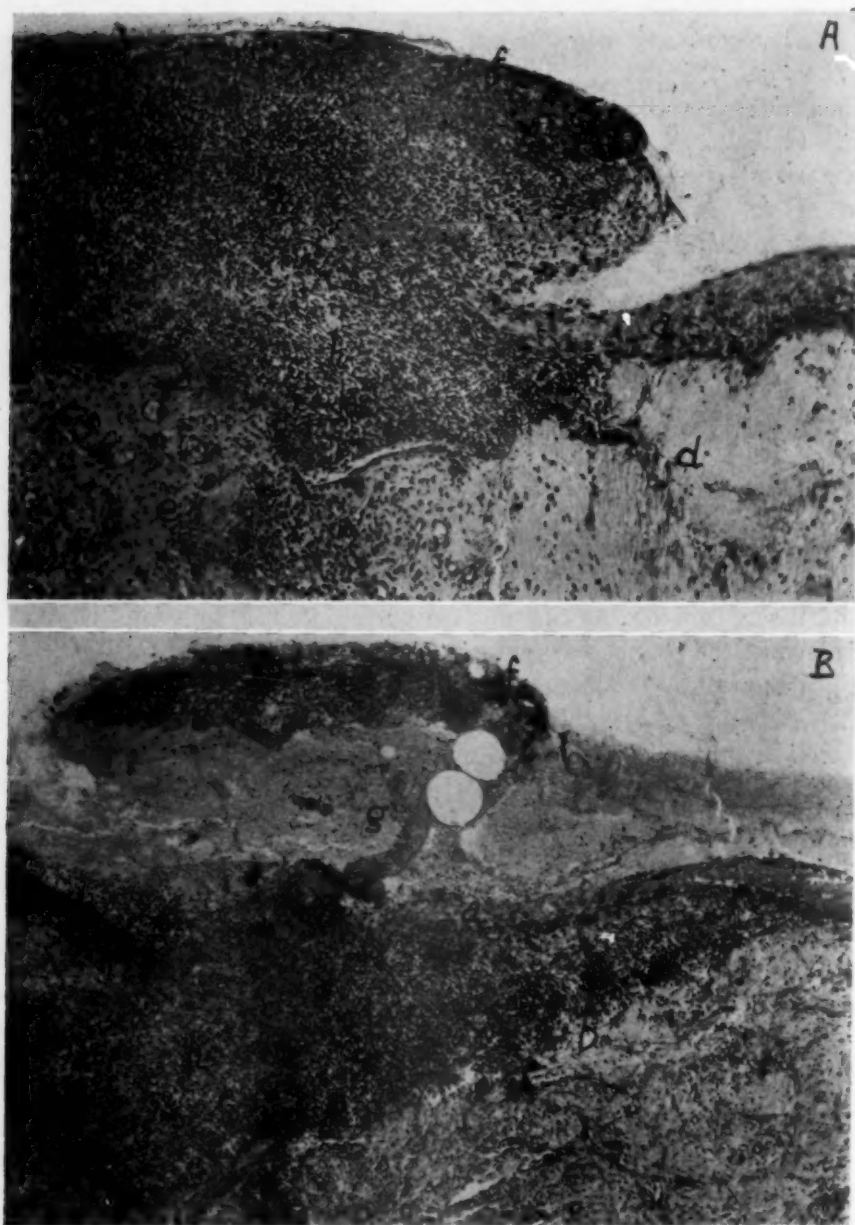


Fig. 3.—Healing process of gingival wound, without postoperative medication. A, two days; B, four days; C, nine days; and D, fourteen days after gingivectomy.

a, Epithelium of gingiva; b, wound surface; c, blood clot; d, edema of connective tissue; e, marked cellular activity in the tissue below the blood clot (necrotic); g, central layer of blood clot (zone of demarcation), h, inner layer of blood clot (fibrinous).



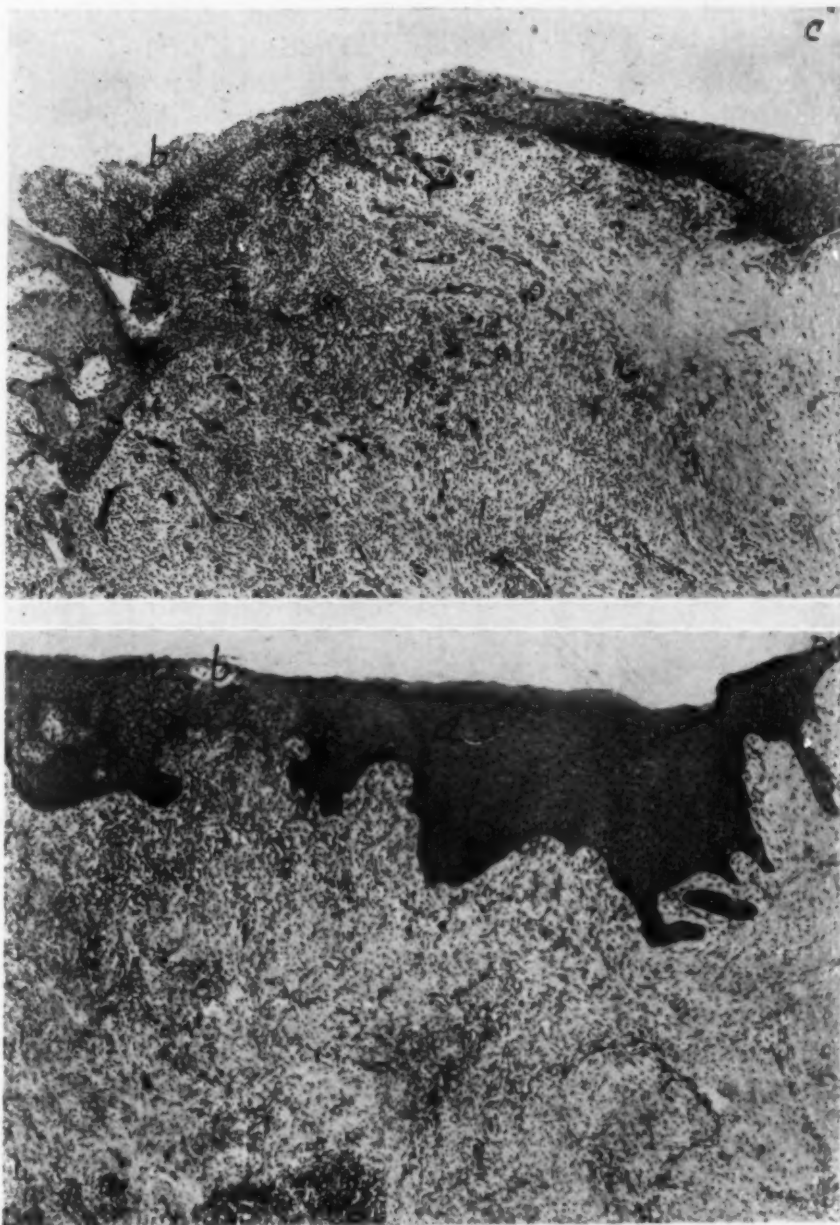


Fig. 3.

(For legend see opposite page.)

nective tissue. The accumulation of leucocytes in the center of the blood clot is a process of demarcation. The function of these cells is to prevent the penetration of bacteria and toxins into the exposed connective tissue. The inner layer of the blood clot is a network of fibrin (*h*). The progressive change of these layers in the blood clot can be seen in the successive specimens. Four days after the first gingivectomy (Fig. 3, *B*) the outer necrotic layer (*f*) of the blood clot has been separated from the rest of the clot by the demarcating leucocytes. Epithelium (*a*) covers a large area of the wound surface. The epithelium, however, does not grow over the original wound surface (*b*), but above the inner layer of the clot which is composed of fibrous network. This inner layer of fibrin becomes organized or transformed into connective tissue. The first evidence of this organization is the penetration of capillaries into the fibrin network. Fig. 5, *A*, shows a section of this fibrin network in which a capillary has been formed by elongated cells. These are most probably endothelial extensions of capillaries from the underlying connective tissue. Red and white blood corpuscles can be seen within this fine, newly formed capillary (*a*).

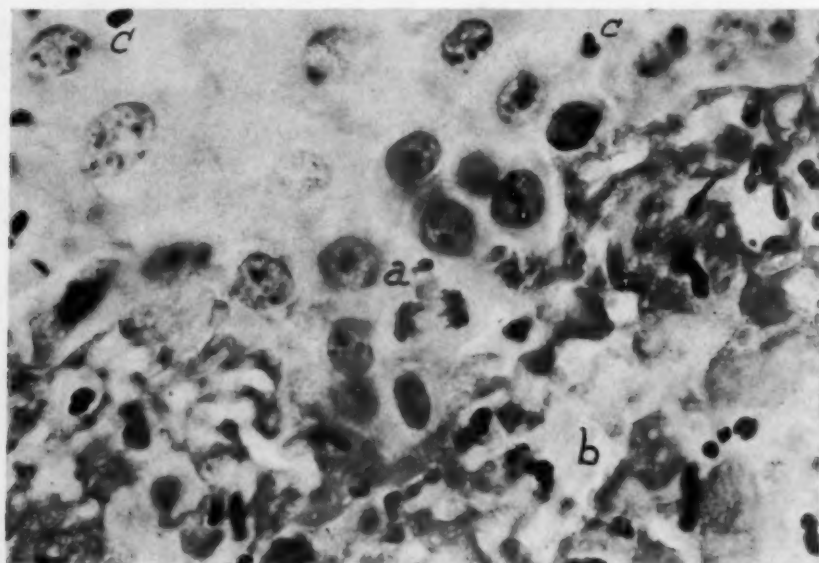


Fig. 4.—High magnification of the epithelium from Fig. 3, *A*. Mitosis in the epithelium (*a*). The cells are round and loosely arranged. Below the epithelium there is a fibrin network (*b*) with leucocytes and a few fibroblasts. Leucocytes migrate through the epithelium (*c*).

While these capillaries are forming, the fibroblasts show abundant mitotic figures (Fig. 5, *B*). As a result of this mitotic activity a large number of new fibroblasts develop in the blood clot (Fig. 5, *C*), thus organizing and utilizing it in the process of tissue regeneration.

Epithelium stretches over the surface of this organizing blood clot and eventually covers it. Students of wound healing claim that shrinkage of the fibrin in the blood clot is a very favorable factor, since it reduces the size of the wound. It is generally accepted, however, that a large blood clot is a hindrance to healing, as it harbors bacteria and prevents rapid epithelization. This has been proved to apply also in gingival surgery. If a large blood clot is left on the surface of a wound, granulating connective tissue may surround it and the clot will remain for a long time within the tissue, thus preventing complete healing.

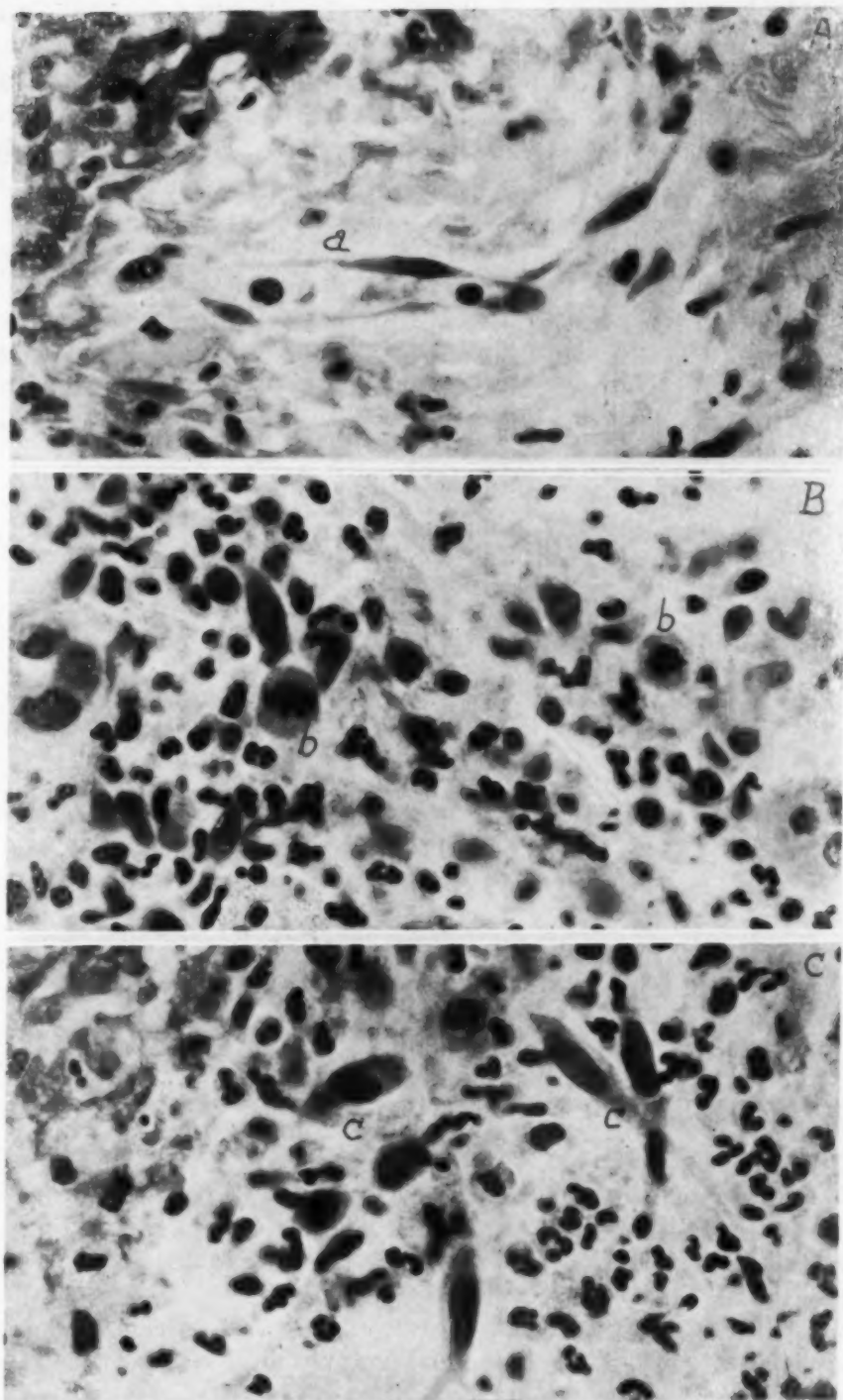


Fig. 5.—Organization of the fibrinous part of the blood clot. A, Formation of new capillaries (a). B, Mitotic activity in the connective tissue (b). C, Development of fibroblasts (c) in the blood clot.



Fig. 6 shows a blood clot (*a*) almost entirely surrounded by granulation tissue (*b*), but twenty-two days after gingivectomy there is still no epithelium covering the surface.

After nine days the tissues show considerable advance in healing, as illustrated in Fig. 3, *C*. There is only a small area free of epithelium which advances on both sides of the wound (*a*). In higher magnification, islands of epithelium are noticeable over almost the entire surface. The free surface is covered by numerous leucocytes (*b*) forming the first line of defense for the exposed connective tissue. Organization of the blood clot is practically complete because only traces of the fibrin network can be recognized. The edema is greatly reduced and a large number of fibroblasts and capillaries are present. The wound seems somewhat elevated above the original line of operation, as there was excessive growth within the connective tissue. This overgrowth is observed clinically as a proliferation of granulation tissue which presents itself as a red, velvety surface, readily bleeding upon touch.

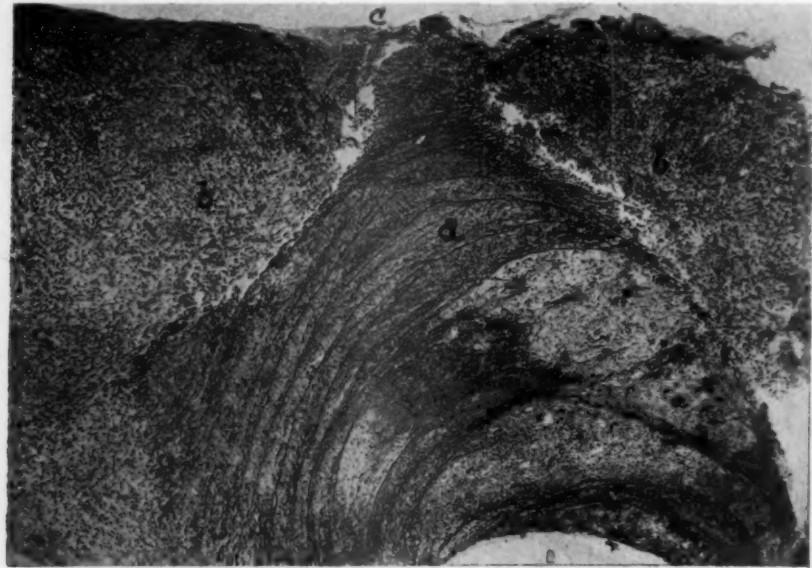


Fig. 6.—Large blood clot (*a*) left on the wound surface, surrounded by the granulating connective tissue (*b*). At one spot the blood clot reaches the surface (*c*).

Fourteen days after the first gingivectomy (Fig. 3, *D*), the epithelial surface has been almost completely regenerated (*a*). There was a microscopically small area where leucocytes penetrated to the surface (*b*). Epithelium was present at this point also, however, and the leucocytes have passed through the epithelial tissue (Fig. 7). Further healing on the surface consists of a functional readaptation of the tissues and proper keratinization. About six weeks after gingivectomy it is difficult to determine, from a biopsy specimen, that an operation had been performed.

The activity in the connective tissue below the wound surface surpasses the activity of the epithelium. The main and most characteristic feature is the change from a chronic inflammatory reaction to an acute condition. The plasma cells which are so characteristic of chronic inflammation of the gingiva (Fig. 1) disappear entirely from the superficial layers. Two days after the operation, it



is difficult to find a plasma cell anywhere in the tissues near the wound surface. Instead of the plasma cells, polymorphonuclear leucocytes, which come from the blood stream, occupy this field. The capillaries are often extremely dilated and filled to capacity with leucocytes, as shown in Fig. 8, *A*. The leucocytes appear a few hours after the operation is performed and their numbers increase up to about the ninth day; from then on, a decrease in their number can be observed.

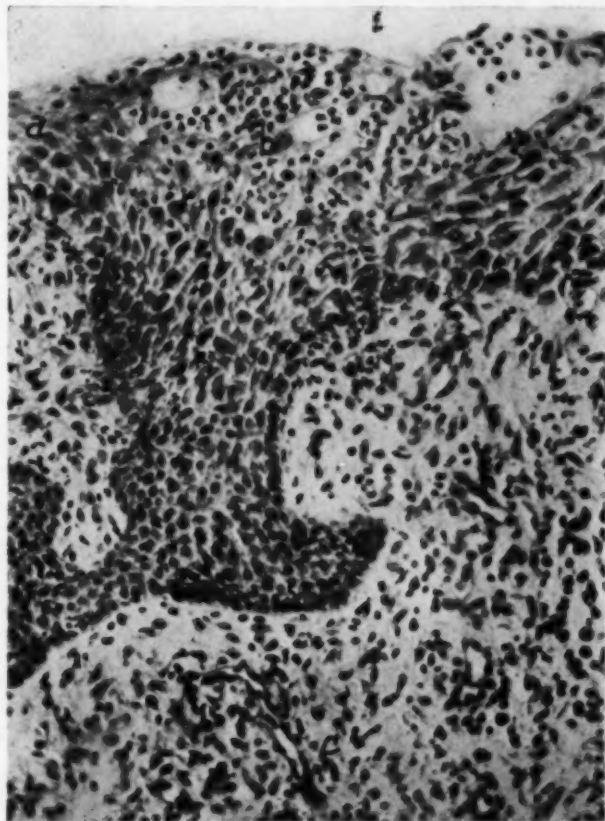


Fig. 7.—Wound surface fourteen days after gingivectomy (high magnification from Fig. 3, *D*). *a*, Epithelium; *b*, leucocytes migrate through the epithelium to the surface; *c*, in the deeper layers the cellular reaction begins to change from leucocytes to lymphocytes and plasma cells.

As the capillaries become engorged with leucocytes, these cells begin to migrate through the walls of the vessels into the surrounding tissues. Fig. 8, *B*, is a higher magnification of Fig. 8, *A*, and shows a leucocyte half in and half out of the capillary (*c*) four days after gingivectomy. Fig. 8, *C*, shows a similar condition on the ninth day. The endothelial cells, which form the wall of the capillaries, are swollen and bulge into the lumen. Numerous polymorphonuclear leucocytes fill these capillaries. The leucocytes in the tissue immediately adjacent to the capillaries frequently take on an elongated shape, indicative of migration. The cause of this migration is discussed extensively in the literature. V. Menkin,<sup>1</sup> one of the outstanding investigators of inflammation, has isolated a product, resulting from the splitting of protein, and has called it "leukotaxin." This seems to originate in inflamed tissues, forming the chemotactic stimulus necessary for the migration of the leucocytes. Shortly before the leucocytes begin to migrate, the endothelial wall of the vessels becomes permeable to blood

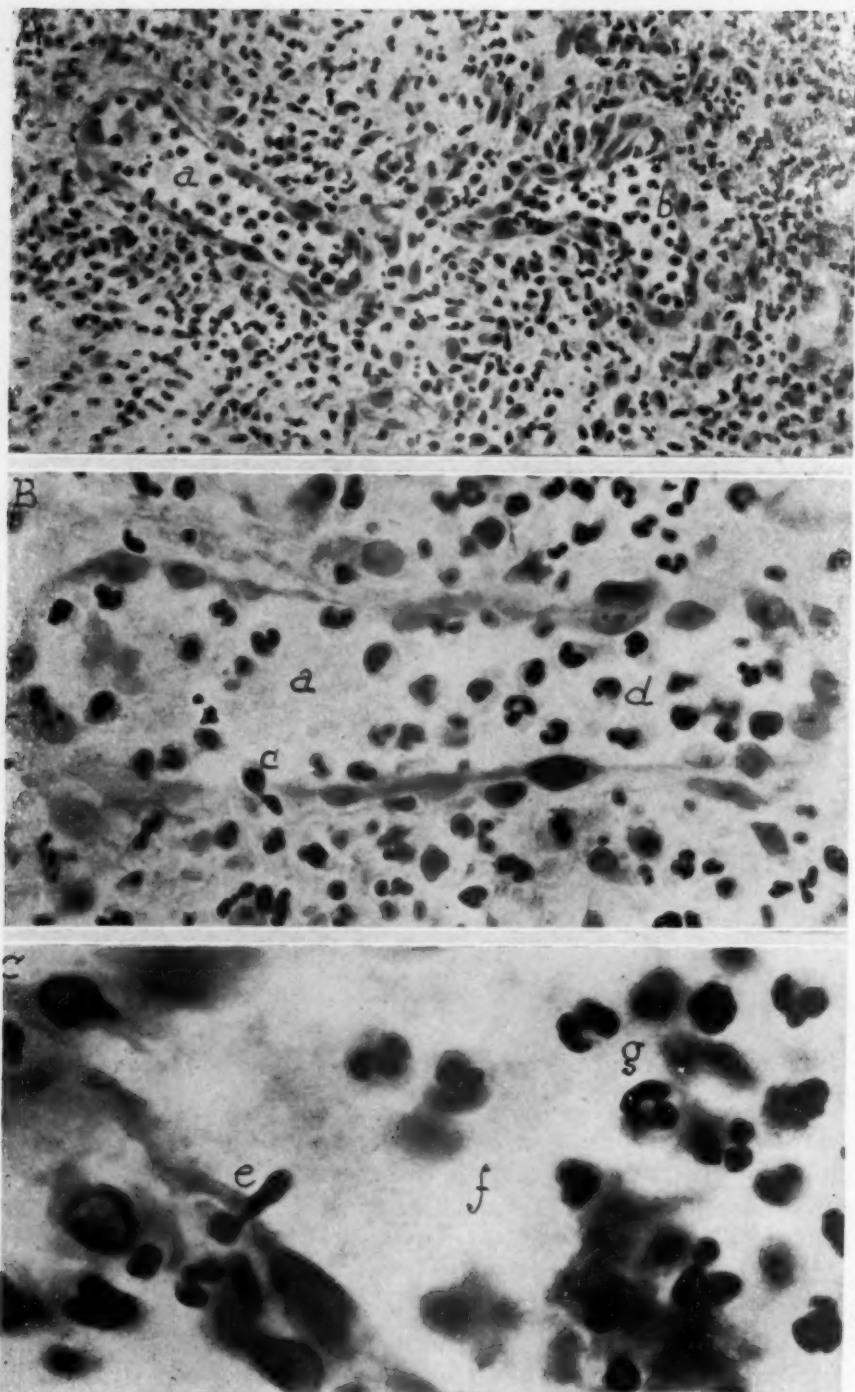


Fig. 8.—A, Leukocytes fill the capillaries (a and b) in the inflammatory area below the wound surface. B, Capillary (a) with migrating leucocyte (c). The lumen of the vessel is filled with leucocytes (d). C, Migrating leucocyte (e) in a capillary (f). Numerous leucocytes (g) in the lumen of the capillary.

plasma, due to toxic influence. Fibrinous fluid accumulates in the tissues surrounding the vessels in the entire inflamed area. The leucocytes migrate toward the surface and their function is phagocytic upon such foreign material as toxins, bacteria, and disintegrating cells, and also to produce or liberate anti-toxins and ferments. A very marked activity begins in the entire connective tissue soon after the start of migration of the leucocytes. Mitotic figures develop in the endothelial cells, producing budlike branches of the vessels (Fig. 9, *A, a*). Due to this multiplication of the endothelial cells a large number of new capillaries develop, thus producing the vascular condition of this granulation tissue.

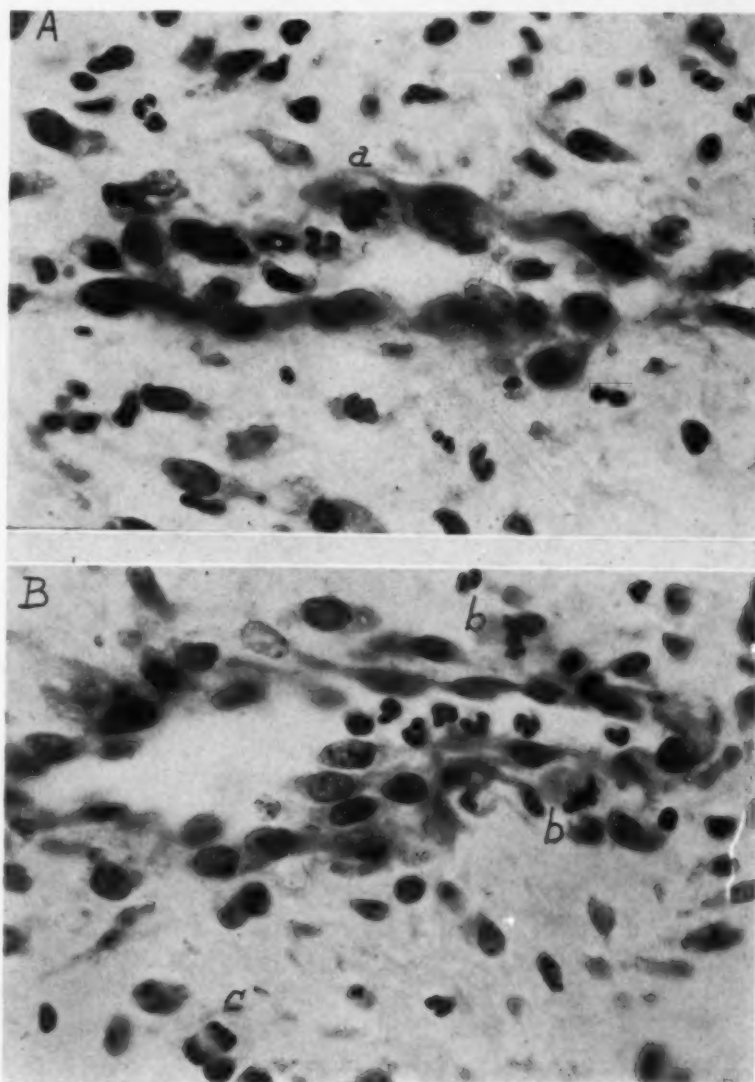


Fig. 9.—*A*, Mitotic activity in an endothelial cell (*a*) of a capillary. *B*, Mitotic activity in the undifferentiated mesenchymal cells (*b*) adjacent to a capillary. Mitosis in a fibroblast (*c*).

A very important feature in wound healing is the multiplication of the undifferentiated mesenchymal cells which lie in close proximity to the capillaries (Fig. 9, *B, b*). They are outside the endothelial wall and, according to

Maximow,<sup>2</sup> carry the embryonic potentiality of developing into any type of mesenchymal cell necessary during the course of the inflammatory reaction. Their presence and activity in inflammation is very important, because they transform into migratory phagocytic elements to aid the healing tissues by their antitoxic-phagocytic capacity.

The fixed connective tissue cells are also very active, as has already been shown in Fig. 5, *B* and *C*. They form new fibroblasts which in turn produce fibers important in walling off the inflammatory process. The acuteness of the inflammatory process diminishes in the deeper layers because of this mesenchymal activity and becomes localized toward the surface.

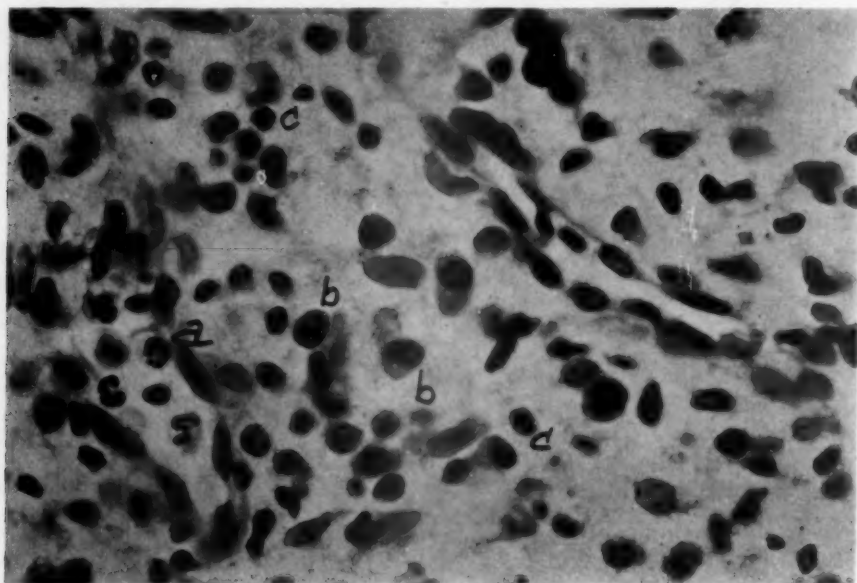


Fig. 10.—Higher magnification of the region (*c*) from Fig. 7 (fourteen days after gingivectomy). Leucocytes are confined largely to the capillaries (*a*). In the connective tissue, plasma cells (*b*) and lymphocytes (*c*) can be seen.

After fourteen days (Fig. 3, *D*) leucocytes can be found only in the epithelium and immediately below it (Fig. 7). Some of the deeper capillaries show leucocytes in their lumen but they do not appear to migrate through the vessel walls (Fig. 10, *a*). The leucocytes have disappeared from around the vessels and nongranular, mononuclear cells have begun to replace them (*b* and *c*). Maximow calls these cells polyblasts, due to their manifold shape and origin. They develop largely from lymphocytes and histiocytes. A few cells have the appearance of plasma cells (*b*) characteristic of chronic inflammation, whereas others are of the small lymphocytic (*c*) type.

The change in the character of inflammation, from acute to chronic, may be due to a change in the pH concentration of the inflamed tissues. V. Menkin claims that, at the beginning of the inflammation, the pH of the tissues is on the alkaline side (approximately 7.2 to 7.4), but during the process of inflammation, acids accumulate as the result of faulty oxidation and turn the pH of the tissues acid (approximately 6.8 to 6.5). It could be assumed that the disappearance of leucocytes and the appearance of lymphocytic elements is due to



this biochemical change. The circulatory disturbances and lack of proper oxidation in the tissues probably also play an important part in this reaction.

The characteristic picture at a certain distance from the healing wound surface is the accumulation of fibroblasts, which, by producing fibers, limit the affected area. Fig. 11, *A*, shows the borderline of inflammation (*a*) and the fibrous capsule (*b*). Close to the fibrous capsule (Fig. 11, *B*) mitotic figures

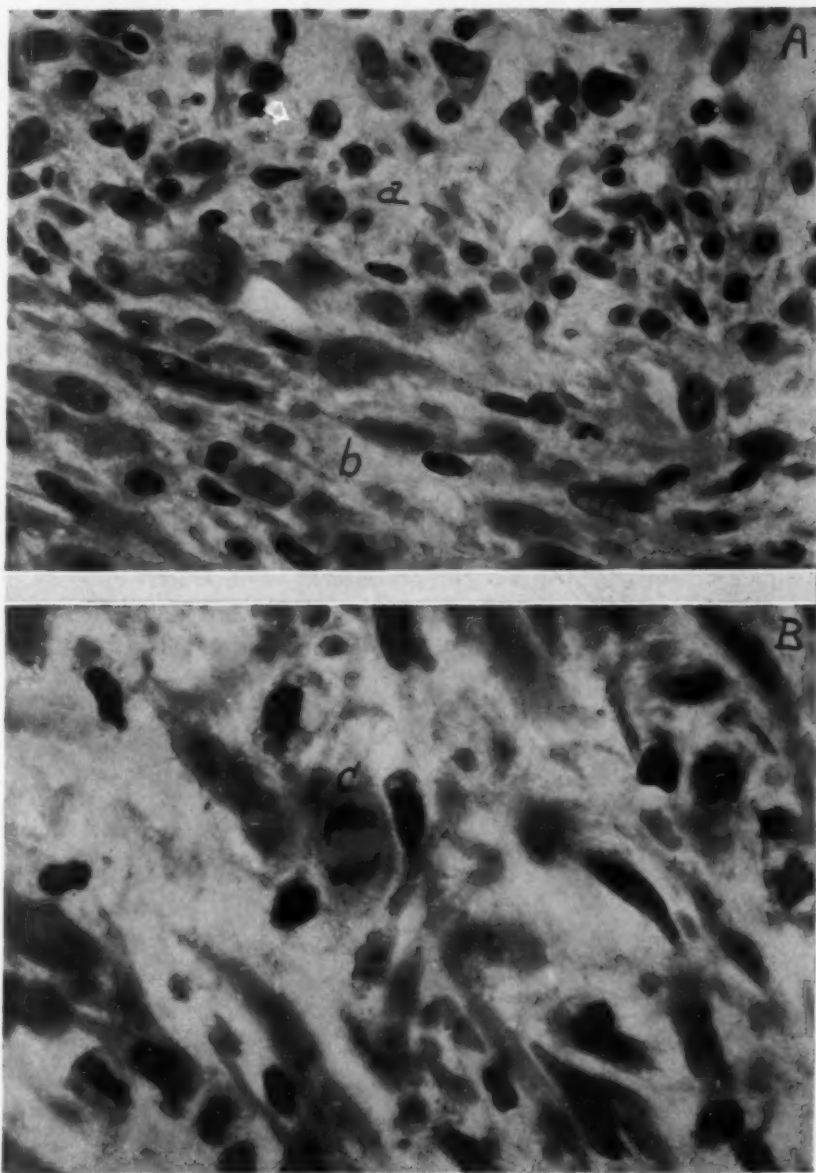


Fig. 11.—*A*, Around the inflammatory area (*a*) a fibrous capsule (*b*) develops, due to multiplication of fibroblasts and fiber formation. *B*, Mitosis of fibroblast (*c*).

(*c*) can be observed in different stages of cell division. Migratory cellular elements are scarce in this region, as fixed fibroblasts and fibers dominate the picture. The acute symptoms of inflammation subside entirely with the complete healing of the wound surface.

## SUMMARY AND CONCLUSIONS

A plan has been devised to study the healing process of the gingival wounds after removal of excessive gingival tissue. This plan consists of removing sections of tissue from the wound surface every two days after the original operation. The process of healing can thus be studied in the microscopic specimens, obtained by successive excisions of tissue.

The healing of wounds, without any postoperative medication after gingivectomy, was studied. The behavior of the blood clot was noted and the progress of re-epithelization of the wound was observed. The reactions of the connective tissue proved to be of great interest. It was noted that following the operation the chronic inflammation of the superficial subepithelial connective tissue turned immediately into acute inflammation. The capillary reaction was the main feature in this reparatory process; the capillaries became permeable for blood plasma, and leucocytes migrated into the tissues. The acuteness of the inflammatory reaction diminished as the healing of the surface of the wound progressed. The epithelial covering of the wound was completed fourteen days after the operation, with increased consolidation of the wound.

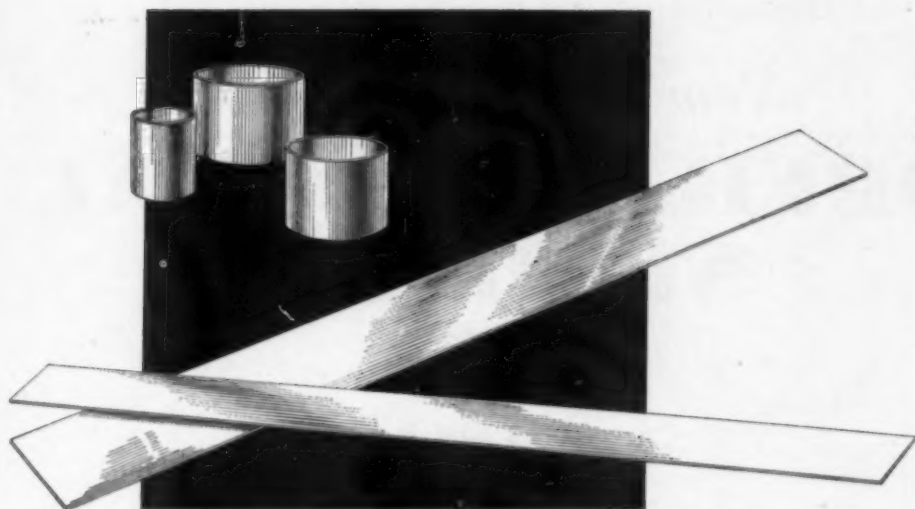
The mechanism of wound healing after gingivectomy is not different from that in other parts of the body. However, the chronic inflammation which is always present in the deeper layers of the diseased gingiva is not influenced by the operation and the consecutive wound healing. This chronic granulation tissue persists after a properly carried out gingivectomy.<sup>3</sup> Surgical removal of all granulation tissue should not be attempted because this would sacrifice tissues which, properly treated, provide the basis for regenerative processes. This granulation tissue, however, should be treated after gingivectomy and by proper treatment can be transformed into an active mesenchymal tissue, capable of regenerative activity.<sup>4</sup> Without proper postoperative medication, the chronic inflammatory tissue will persist and will sooner or later lead to recurrence of pockets.

Gingivectomy alone in itself is only one phase in the treatment of the inflamed gingiva; it is a method of eliminating the unhygienic gingival pocket and thus of creating favorable topographic conditions for the healing of the chronic gingivitis.

The chronic inflammatory condition of the gingiva might also be responsible for the frequently observed excessive granulation of the wound surface following gingivectomy. The excessive granulation of the wound surface would lead to reformation of the gingival pocket and thus upset the aim of the operation.<sup>5</sup> By proper postoperative medication this excessive granulation can be checked; therefore, proper postoperative treatment after gingivectomy is imperative.

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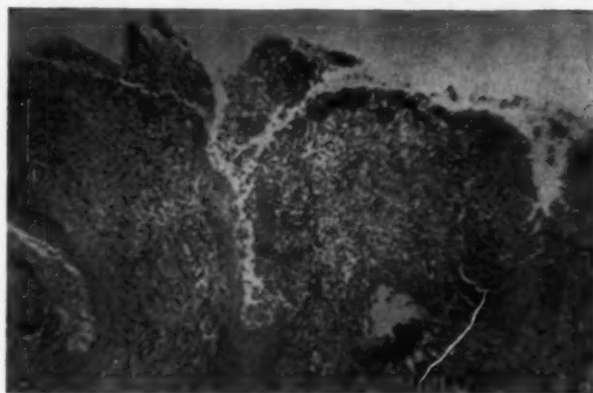
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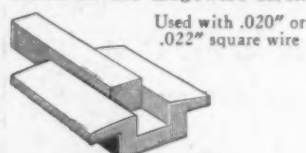
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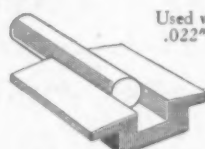
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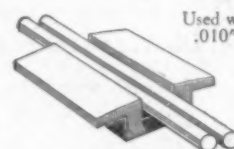
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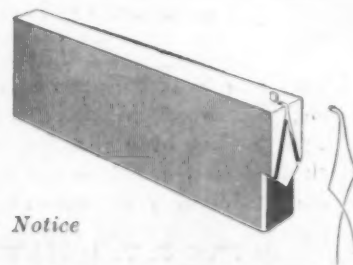
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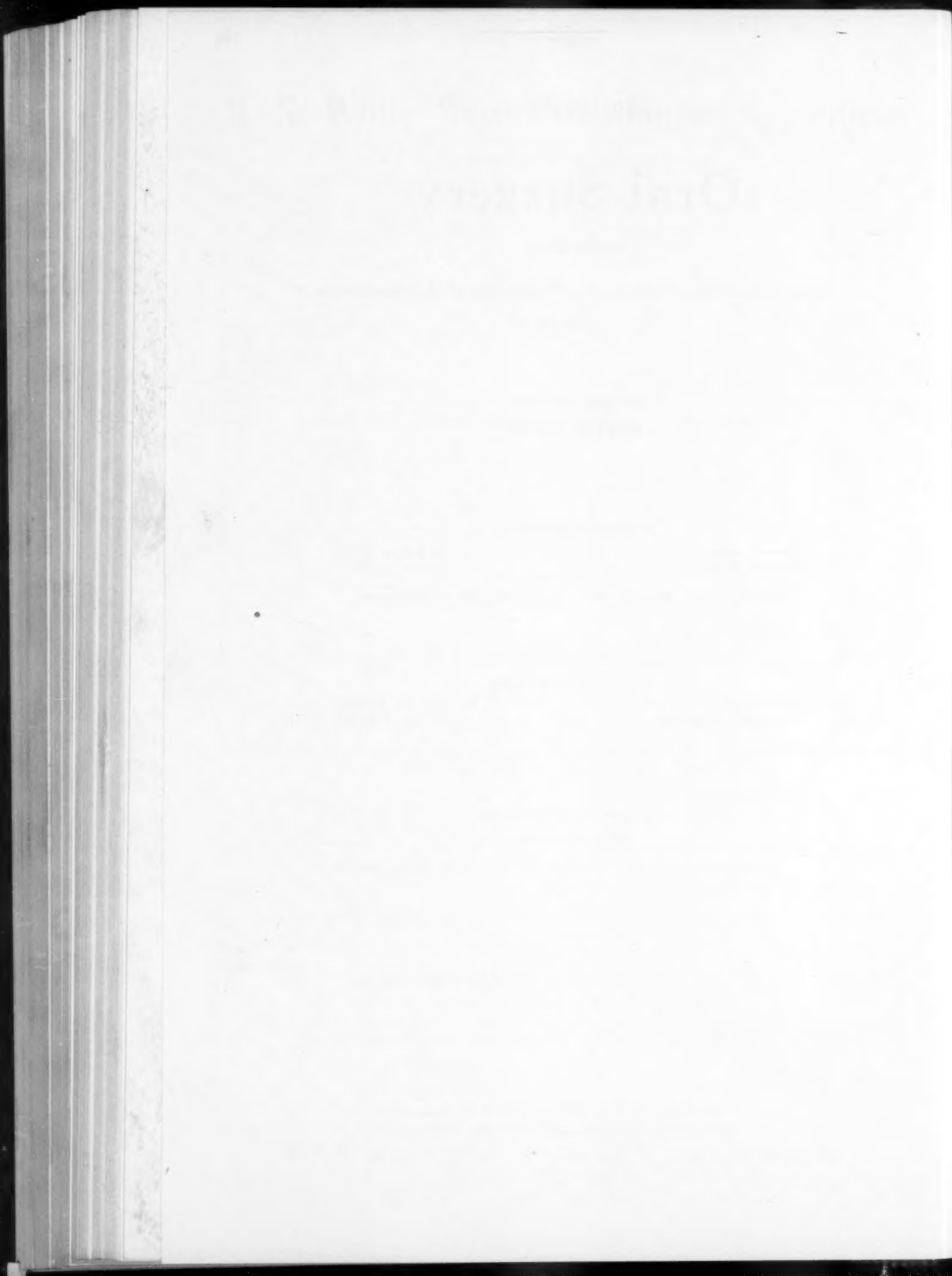
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## Original Articles

### SUBACUTE BACTERIAL ENDOCARDITIS AND DENTAL PROCEDURES

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#### INTRODUCTION

MANY clinicians have suggested that there may be a relation between dental infection and systemic disease. In 1809 Benjamin Rush<sup>1</sup> stated that he had "only added to the observations of other physicians in pointing out a connection between the extraction of decayed and diseased teeth and the cure of general disease."

It was not until the twentieth century that laboratory and clinical evidence regarding the effect of dental infection on systemic disease was studied. Early in the present century the concept of "focal infection" was developed by Rosenow and Billings, and infected teeth were considered one of the important foci.

Among the systemic diseases thought to be caused by dental infection was subacute bacterial endocarditis. Many writers, including Thayer,<sup>2</sup> Weiss,<sup>3</sup> Christian,<sup>4</sup> and Blummer,<sup>5</sup> have cited infected teeth as a portal of entry for the organism causing endocarditis. It has also been observed that the extraction of teeth has been closely followed by subacute bacterial endocarditis.

Certain aspects of the connection between dental infection and the systemic diseases, subacute bacterial endocarditis and leucemia, are not clear. For example, while most discussions of subacute bacterial endocarditis following dental extraction conclude that the extraction caused the endocarditis, it must not be overlooked that subacute bacterial endocarditis has an insidious onset, and, in the cases reported, these symptoms may have preceded extraction. While there is evidence that subacute bacterial endocarditis may follow extraction, it has been stated, on the other hand, that the retention of infected teeth is potentially dangerous to individuals with valvular damage. There remains, then, the question as to whether or not extraction should be performed in persons with valvular damage. The bearing that the type of dental anesthetic and the technique of extraction has on the likelihood of resulting endocarditis is uncertain.

#### LITERATURE

1. *Literature Concerning the Presence of Streptococci in and Around the Teeth.*—Since the first section of the study concerns the relation of the extrac-

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tion of teeth to subacute bacterial endocarditis, some mention of the nature of subacute bacterial endocarditis must be made. Likewise, because of the close relation between endocarditis and rheumatic fever, something concerning the nature of rheumatic fever must be reviewed.

Subacute bacterial endocarditis is a disease in which thrombi containing *Streptococcus viridans* form on the endocardium. Certain conditions greatly increase the susceptibility of the endocardium to these formations.<sup>6</sup> In this connection, Von Glahn and Pappenheimer<sup>7</sup> have suggested that valves with recent or active vegetations are particularly liable to infection by *Str. viridans*. Another predisposing condition is malformation of the valves.<sup>8</sup> A third condition, which has been emphasized by Christian,<sup>9</sup> is the absence of auricular fibrillation.

Osler<sup>10</sup> is usually credited with the first description of subacute bacterial endocarditis (1885). Of ten cases reported by him, there is reason to believe that the portal of entry of the causative organism was by way of the respiratory tract in two cases.

That the portal of entry of *Str. viridans* is sometimes by way of the respiratory tract was pointed out in 1909 by Thomas Horder<sup>11</sup> who referred to the "undoubted fact that the source of the infective agent in most cases is the mouth or intestine," and suggested that attention might be directed profitably to these regions. Billings<sup>12</sup> insisted that the teeth, tonsils, and other cryptic structures were portals of entry for streptococci which were responsible for systemic diseases. Rosenow<sup>13</sup> maintained that different types of streptococci had a predilection for localization ("elective affinity") on different tissues. While the work of Billings and Rosenow has been discredited, Holman,<sup>14</sup> in an apparently unbiased evaluation, stated that the concept of foci of infection is a principle of bacteriology. Hatton<sup>15</sup> believed that there was enough evidence in favor of Billings' stand for clinicians to take into consideration the dissemination of bacteria from infected teeth.

Christian stated that *Str. viridans* causing subacute bacterial endocarditis is the same organism that is found in the dental pulps and around the teeth, which serve as portals of entry. Blummer<sup>16</sup> stated that in cases under his observation *Str. viridans* were isolated from the blood stream at the same time they were present around the roots of teeth. Fulton and Levine<sup>17</sup> suggested that extraction precipitated the development of subacute bacterial endocarditis. Thayer<sup>18</sup> believed that periodontal sepsis was one of the ways by which streptococci gain entrance to the blood stream, and pointed out that 40 per cent of his 77 cases had pyorrhea or periapical involvement.

The incidence, reported by Smith and Brumfiel,<sup>19</sup> of "oral sepsis as a predisposing cause" of subacute bacterial endocarditis is in agreement with Thayer's figures. Of 28 cases reported, "oral sepsis" was found ten times. Weiss<sup>3</sup> stated that the upper respiratory tract was the most common portal of entry of *Str. viridans* and that the teeth constituted a portal of entry.

The first symptoms of subacute bacterial endocarditis are those of most infectious diseases; malaise, chills, fever followed by weakness, loss of weight, and embolic phenomena are frequent.

The etiology of rheumatic fever is unknown; there is considerable evidence that it is a disease in which the streptococcus plays a part. Paul, Salinger, and



Zuger<sup>20</sup> have pointed out that a streptococcal infection, such as a pharyngitis, activates pre-existing rheumatic endocarditis, and that the interval of time between the streptococcal infection and active rheumatic endocarditis is from one to three weeks. The interval supports the hypothesis that a rheumatic endocarditis is injury from an immunologic response of hypersensitive tissue to bacterial antigen. Recently, Rich and Gregory<sup>21</sup> produced in rabbits subjected to experimental serum sickness lesions which resembled Aschoff bodies. The writers pointed out that human serum sickness and rheumatic fever have in common several characteristics. Von Glahn and Pappenheimer argued that it is the active rheumatic vegetation to which the thrombus containing the *Str. viridans* adheres, and that the dissemination of streptococci into the blood during the active phase of chronic rheumatic endocarditis is particularly dangerous to the patient.

While Rush, in 1818, suggested that dental infection was responsible for some systemic diseases, evidence of this was not forthcoming until the work of Billings, Rosenow, and Jackson, which started in 1912. A discussion of Rosenow's work in support of his concept of foci of infection, in which the teeth were among the septic foci, cannot be undertaken and would be out of place in this paper. Billings, Rosenow, and their followers believed that the organisms around the apices of infected teeth were disseminated by the blood, and produced infections at other sites, including the endocardium. In 1915 Rosenow<sup>13</sup> stated that there existed affinity between the various streptococci and the tissues of the body. His evidence for this was that streptococci which had produced a certain lesion in man would, in a high percentage of cases, produce the same type of lesions in rabbits. The work of Billings and Rosenow is discredited because it has not been confirmed by other workers, and because diseases such as poliomyelitis and peptic ulcer, which Rosenow believed to be of streptococcal origin, are not of bacterial origin.

Referring to dental caries rather than to dental infection in general, Arnett and Ennis<sup>22</sup> in 1933 presented evidence against a relation between caries as revealed by x-ray findings and systemic disease. On the basis of 883 routine dental and medical examinations, there was no relation between changes seen in the roentgenograms and systemic disease.

If the concept that the teeth act as "foci of infection" is tenable, streptococci must be found around the apices or in the pulps of teeth in a considerable number of cases. Evidence on this point has been presented by Lazarus-Barlow,<sup>23</sup> Henrici and Hartzell,<sup>24</sup> Haden,<sup>25</sup> Deacon and Swartz,<sup>26</sup> Rhoads and Dick,<sup>27</sup> and Tunnicliff and Hammond.<sup>28, 29</sup> Bacteriologic examination of the apical pulp was carried out in these investigations, excepting that of Lazarus-Barlow, by extracting the tooth carefully, using precaution to prevent oral contamination of the root. Material for a culture from the pulp was obtained by sterilizing the surface of the tooth and either cracking it open or drilling into the tooth. Lazarus-Barlow cultured from alveolar bone removed at autopsy.

Evidence gradually accumulated that all of the bacteria being found in the pulps of teeth were not present naturally. In 1936, Fish and Maclean<sup>30</sup> stated their belief that the pumping action resulting from the extraction of teeth might force the bacteria from the apex into the capillaries of the pulp. The following year Burket and Burn<sup>31</sup> proved the correctness of this idea by show-

ing that a nonpathogen (*Serratia marcescens*) placed in the gingival crevice could be demonstrated at the apex of the tooth and within the pulp, and for a short time, in the blood stream following extraction of the tooth.

In 1938 Kanner<sup>32</sup> showed how bacteria may enter the pulps of intact teeth by an in vitro experiment. An atmospheric pressure within the pulp chamber of a tooth slightly lower than on the surrounding culture media forced the bacteria into the ruptured capillaries of the pulp. The approach, followed by Hayes,<sup>33</sup> to the problem of the bacterial flora of pulps is not subject to the comment that the bacteria may have been pumped into the capillaries by extraction movements of the teeth. *Str. viridans* was isolated 53 times from 340 pulp canals undergoing treatment.

Perhaps Burket's study<sup>34</sup> might be selected as the best and most recent evidence of the presence of *Str. viridans* at the apices of teeth. The periapical areas of 419 teeth were analyzed by cutting through the cortical plate of the alveolar process and obtaining material for culture. Of the 206 positive cultures, *Str. viridans* was found 127 times in either pure or mixed culture.

Using the technique of removing anterior teeth in situ, Burket<sup>35</sup> found that cultures of 54 apices with the contained pulp resulted in growth of *Str. viridans* 16 times. Cultures of the periapical region of the same teeth yielded *Str. viridans* 12 times. Two of the 54 teeth had exposed pulps and none were frankly loose.

The findings of Gunter, Appleton, Strong, Reader, Zimmerman and Brooks<sup>36</sup> are contrary to those of Burket with regard to the bacteriology of the vital dental pulp. These workers chose seventeen vital teeth and isolated each from the oral cavity by a rubber dam. Cultures were taken from the pulps after exposing them with sterile burrs. Only one pulp yielded a positive culture; the organism isolated was not mentioned. After extraction of the teeth, in such a manner as to avoid contamination by saliva, the apices were cultured with uniformly positive results. While this evidence is not in agreement with that produced by Burket's work on the bacteriology of the dental pulp, it supports the work of Burket and of Kanner with regard to the introduction of bacteria into the capillaries of the pulp from the gingival crevice by extraction movements.

2. *Literature Concerning the Occurrence of Bacteremia Following the Extraction of Teeth.*—It has been known for some time that a bacteremia sometimes follows operative procedures, and in 1932 Richards<sup>37</sup> demonstrated that the massage of inflamed gingiva would produce a transitory bacteremia. Other workers have confirmed Richards' findings. The reports are summarized in Table I.

It is apparent that a remarkably large percentage of the blood cultures taken soon after extraction showed streptococci. There is good evidence that the positive cultures represent true bacteremias and not contaminants. A positive culture was obtained from only one member of Burket and Burn's "control group." All but one of the pre-extraction cultures made by Faillo were negative and all were negative in Palmer and Kempf's series. The skin was tested for sterility by Murry and Moosnick and no growths were obtained. Further evidence that the venipuncture was not responsible for the positive cultures is found in the results when subsequent cultures of blood taken from the same in-

dividuals and in the same manner were made. Repeated samples were taken to determine the duration of the bacteremia that followed extraction. Samples taken an hour or more after the extraction were sterile.

The reason for the great variation in the number of individuals with streptococcal bacteremias resulting from extraction is not apparent. There are three factors that seem to determine the probability of bacteremia: the presence or absence of pyorrhea, the type of anesthetic used, and the severity of the manipulation. It is not possible to state whether these three factors are the chief ones, or what might be the role of each. The clearing power of the blood for bacteria is integral with the natural immunity of the individual. Possibly streptococci are present in the blood more frequently than indicated, but because of vigorous antibody activity, the organisms are not detected in a culture taken several minutes after extraction.

The streptococci present in a bacteremia may come from the periapical or the periodontal region, or the pulp. As pointed out by Burket and Burn, and by Faillo, efforts directed at sterilizing the periodontal tissues will reduce the incidence of bacteremia following dental extraction.

That the bacteremia following dental manipulation is transitory is evidenced by the findings of those who took subsequent cultures. The blood was sterile in from ten minutes to eight hours in O'Kell and Elliott's series, and after six hours in every case in Faillo's series.

The transitory dissemination of bacteria into the blood stream which results from dental manipulation is a mechanism that may cause infectious disease and injury resulting from allergic reaction. There is evidence that this entry of bacteria occasionally is responsible for subacute bacterial endocarditis and active rheumatic endocarditis.

3. *Literature Concerning Subacute Bacterial Endocarditis and Dental Procedures.*—In reviewing reports of subacute bacterial endocarditis following the extraction of teeth, some evaluation has been attempted. Before a report is accepted as one of subacute bacterial endocarditis resulting from the removal of a tooth, it must be determined with reasonable certainty that:

1. The patient had subacute bacterial endocarditis. It has been pointed out (Osler, Blummer, Horder, Von Glahn) that subacute bacterial endocarditis may be confused clinically with acute rheumatic endocarditis and with chronic endocarditis.

2. The onset of subacute bacterial endocarditis followed extraction.

3. The first symptoms which followed extraction were those of subacute bacterial endocarditis.

4. The interim between extraction of the tooth and the onset of subacute bacterial endocarditis was compatible with the hypothesis that bacteremia from the extraction of the tooth produced the endocarditis.

While a number of authors, Horder,<sup>11</sup> Stader,<sup>45</sup> Blummer,<sup>5</sup> and Smith, Sauls, and Stone,<sup>46</sup> have referred to subacute bacterial endocarditis following dental infection without describing a specific case, a number of reported cases were found. These have been analyzed and placed in four groups. In the first group there are fifteen reports in which there is reasonably conclusive evidence that subacute bacterial endocarditis resulted from dental extraction. These reports are summarized in Table II.

TABLE I  
STUDIES OF BACTEREMIAS RESULTING FROM DENTAL MANIPULATIONS

YEAR	AUTHOR	DESCRIPTION OF DENTAL CONDITION OF THE GROUP AND MANIPULATION INVOLVED	TYPE OF ANESTHETIC	NO. IN GROUP	NO. WITH POS. CUL.	PATIENTS POS. FOR STREP. VIRIDANS			COMMENTS
						NO.	% OF ENTIRE GROUP		
1932	Richards <sup>37</sup>	"Definitely inflamed gums." Massage of gums	None	17	3	The organism isolated not stated			All cultures before extraction were negative
1935	O'Kell, Elliott <sup>38</sup>	Divided 138 patients into 3 groups on basis of amount of pyorrhea present: Group A Marked pyorrhea. Extraction Group B Moderate pyorrhea. Extraction Group C "No noticeable pyorrhea." Extraction	General General General	40 60 38		30 42 12	75 70 34		Cultures from control group of 30 individuals were all negative. Certain strains of the streptococci isolated from the blood after extraction were serologically identical with the organisms causing endocarditis lenta in other patients.
1936	Round, Kirkpatrick, Hails <sup>39</sup>	Five exhibited alveolar absorption; condition of other 5 not described. Masticated hard candy	None	10	2	1	10		Cultures from areas of venipunctures taken on all 10 patients were negative
1937	Burket, Burn <sup>31</sup>	Group 1 Random sampling from clinic admissions. Extraction	Local	53	12	0	0		A test organism, <i>S. marcescens</i> was painted in the gingival crevice. This organism was isolated from blood after extraction. Iodine was painted in gingival crevice before extraction



	Group 2 Same as Group 1	Local (86) N <sub>2</sub> O (1) None (5) Local	92	11	4	4.3	This group received no treatment of gingival crevice prior to extraction
1938	Palmer, Kempf <sup>40</sup> with Jos. A. Hopkins	Local	82	14	11	13.4	Pre-extraction cultures were made on every patient. All were negative.
1939	Elliott <sup>41</sup>	Not stated	21	Not stated	18	86	
1941	Murry, Moosnick <sup>42</sup>	None	336	185			Cultures from skin at site of venipuncture were consistently negative. In 84% of patients organisms isolated were a "small diplococci;" further identification not reported
1942	Faillio <sup>43</sup>	Local	20	9	Not stated		Pre-extraction cultures were made on all patients; all but one were negative
1943	Northrop, Crowley <sup>44</sup>	Local	93	12	6	6	All but one culture was negative within 10 min. after extraction

TABLE II  
REPORTED CASES OF SUBACUTE BACTERIAL ENDOCARDITIS RESULTING FROM EXTRACTION OF TEETH

NO. YEAR	AUTHOR	AGE	SEX	STATUS OF ENDOCARDITIS BEFORE EXTRACTION	INTERVAL	FIRST SYMPTOM OF SUBACUTE BACTERIAL ENDOCARDITIS	EVIDENCE OF SUBACUTE BACTERIAL ENDOCARDITIS	COMMENTS AND CONCLUSION
1 1926	Thayer <sup>2</sup> Case 33M45941	28	M	Apparently had recovered from recent attack of rheumatic fever. Had gained 6 pounds	Not stated	Fever, sweating, fatigue, "symptoms of bacterial endocarditis"	Positive culture of blood. Subacute bacterial endocarditis of mitral valve	Probably subacute bacterial endocarditis resulting from postextraction bacteremia. Thayer believed the endocarditis resulted from extraction
2 1930	Rushion <sup>47</sup> Case 1	26	F	No evidence of previous valvular damage	Indefinite. Excessive bleeding followed extraction	Record indefinite. Weakness, depressed, malaise, followed by pleurisy, and night sweats	"Confirmed at autopsy"	Unable to determine interim between extraction and onset of symptoms. Probably extraction caused the subacute bacterial endocarditis
3	Case 2	6	M	History of rheumatic fever at 4 years of age. Apparently inactive at time of extraction	Three days	Fever of 102.6° F. Blood positive for <i>Str. viridans</i> 9 days after extraction	Osler's nodes. Hemiplegia	Subacute bacterial endocarditis following postextraction bacteremia and on previously damaged valve
4 1931	Abrahamson <sup>48</sup> Case 1	39	M	"Well compensated mitral regurgitation." No symptoms	"Shortly"	"Small red spots on his fingers"	Petechiae, blood positive for <i>Str. viridans</i> . "Tender nodules" at various sites on body	Good evidence that subacute bacterial endocarditis resulted from extraction. Some evidence that active rheumatic fever followed extraction

5	1932	Bernstein <sup>49</sup>	25	M	History of rheumatic fever at 8 years of age. No evidence of rheumatic activity at time of extraction	Alveolar bleeding almost immediately. Fever on 5th postoperative day	Fever, excessive bleeding from dental alveolus, leucocytosis. Blood positive for <i>Str. viridans</i> 26 days after extraction	Positive culture from blood, Osler's nodes, anemia, petechiae	Subacute bacterial endocarditis resulting from postextraction bacteremia
6	1933	Vanderhoof <sup>50</sup> Case 1	56	M	Apical systolic murmur. Dyspnea on exertion. Teeth extracted because of abscesses. No evidence of rheumatic activity at time of extraction	Four days	Malaise	Petechiae. Culture of blood positive for <i>Str. viridans</i>	Case of subacute bacterial endocarditis caused by postextraction bacteremia
7		Case 2	19	F	History of rheumatic fever as a child. No evidence of activity at time of extraction	Within 12 hours. Great difficulty in removing tooth	Chill, followed by fever which continued. "Presented typical picture of subacute bacterial endocarditis"	Culture of blood positive for <i>Str. viridans</i> . Petechiae. Autopsy confirmation	Case of subacute bacterial endocarditis caused by postextraction bacteremia. Chill explained on basis of bacteremia
8		Weiss <sup>53</sup> Case 5	31	M	Acute rheumatic fever 7 years before extraction. Teeth extracted to relieve arthralgia present	Three weeks	Fever and sweats followed by red spots on finger tips	Petechiae. Blood yielded <i>Str. viridans</i> . Café au lait complexion. Hematuria. No autopsy	Subacute bacterial endocarditis resulting from extraction occurring on valve with active rheumatic endocarditis
9		Weiss	24	M	Hospitalized 2 years before extraction because of acute rheumatic fever. No evidence of activity at time of extraction	Tooth first treated, then extracted. Two weeks later first signs appeared	General weakness, malaise, pallor, which increased; septic fever followed	Petechiae. Blood positive for <i>Str. viridans</i>	Not entirely clear that there was no active endocarditis at time of extraction. Probably case of subacute bacterial endocarditis resulting from dental extraction

TABLE II—CONT'D

NO.	YEAR	AUTHOR	AGE	SEX	STATUS OF ENDO-CARDITIS BEFORE EXTRACTION	INTERVAL	FIRST SYMPTOM OF SUBACUTE BACTERIAL ENDOCARDITIS	EVIDENCE OF SUB-ACUTE BACTERIAL ENDOCARDITIS	COMMENTS AND CONCLUSION
10		Case 10	33	F	History of chronic endocarditis. Active rheumatic fever at time of extraction. As stated by Weiss	Not stated	Temperature of 100-102° F. Shaking chill, loss of weight	Blood positive for <i>Str. viridans</i> . Hematuria. Infarct of spleen. No autopsy	Probably subacute bacterial endocarditis on valve which was site of active rheumatic endocarditis. Impossible to ascertain if symptoms at time of extraction were subacute bacterial endocarditis or active rheumatic fever
11		Feldman <sup>31</sup> Case 2	22	F	History of rheumatic fever. No evidence of active endocarditis of any kind at time of dental extraction	Not stated exactly. Short	Chills and fever	Petechiae. Blood positive for <i>Str. viridans</i> . Café au lait complexion. Duration of illness compatible with subacute bacterial endocarditis. No autopsy	Subacute bacterial endocarditis probably resulting from dental extraction
12		Case 4	24	F	History of "heart trouble" during childhood. Was in "perfect health" at time of extraction	Ten days	Loss of strength, chills	Blood positive for nonhemolytic streptococcus. Splenic and renal infarcts. Café au lait complexion	Case of subacute bacterial endocarditis resulting from extraction of abscessed tooth



13	Case 3	18 F	History of rheumatic endocarditis at age of 8 years. Health as "well as usual" at time of extensive dental work. No extraction done	"Within a few days"	Chills, fever, night sweats, Osler's nodes	Blood positive for <i>Str. viridans</i> . Vegetation on mitral valve	Dental manipulation caused bacteremia which resulted in subacute bacterial endocarditis
14	1942 Budnitz, Nizel, Berges	25 M	History of rheumatic heart disease at 2 years of age. "Perfect health" at time of extraction	Not indicated	Chills, fever, malaise sufficient to confine the patient to bed	<i>Str. viridans</i> cultured from vegetation on tricuspid and pulmonary valves. Infarcts of spleen focal embolic glomerulonephritis	Subacute bacterial endocarditis probably resulting from extraction. Conclusive evidence lacking because interim between extraction and first symptom of subacute bacterial endocarditis not stated.
15	1942 Geiger <sup>53</sup>	34 M	History of 3 attacks of rheumatic fever. No evidence of active rheumatic fever at time of extraction	About 1 week	Chronic fatigue. Pericardic palpitation of heart, progressive loss of weight, positive culture of <i>Str. viridans</i> 60 days later	Subacute bacterial endocarditis of valves, emboli. <i>Str. viridans</i> cultures from root canals and periapical abscesses as well as vegetations	Subacute bacterial endocarditis probably result of post-extraction bacteremia

TABLE III  
RHEUMATIC ENDOCARDITIS BECOMING ACTIVE FOLLOWING EXTRACTION OF TEETH

NO.	YEAR	AUTHOR	AGE	SEX	STATUS OF ENDOCARDITIS BEFORE EXTRACTION	INTERVAL	FIRST SYMPTOM OF SUB-ACUTE BACTERIAL ENDOCARDITIS	EVIDENCE OF SUBACUTE BACTERIAL ENDOCARDITIS	COMMENTS AND CONCLUSION
16	1936	Rushton <sup>47</sup> Case 3	10	M	Apparently recently recovered from active rheumatic fever	One month	Pain in limbs thought to be rheumatic fever. Diagnosis of subacute bacterial endocarditis was made later	None except that diagnosis of subacute bacterial endocarditis was made	Not Rushton's own case, reported from a Dr. French. Probably active rheumatic fever resulting from extraction. The interim of one month is compatible with this conclusion

Rushton's third reported case occupies a singular position among the reports, and has been placed in Table III. There is evidence that in this case the dental extraction caused chronic rheumatic endocarditis to become active. This is the only instance of the kind found in the literature with the possible exception of Report 4 in Table II.

Table IV sets forth the pertinent facts from ten reports in which dental extraction *probably* caused subacute bacterial endocarditis. As indicated by the comments and conclusions regarding the reports in Table IV, the evidence submitted in Reports 17, 22, 23, and 26 that the patient had subacute bacterial endocarditis rather than some other cardiac disease was not conclusive. Other cases are in this group because it was not clear that infective endocarditis did not exist at the time of extraction.

Table V is composed of fourteen reports of subacute bacterial endocarditis which *may* have resulted from extraction but in which not enough evidence was published to make possible an accurate evaluation. There is insufficient evidence to substantiate the diagnosis of subacute bacterial endocarditis in Reports 27, 28, 30, 31, 32, 33, 34, 35, 36, and 37. That the bacteremia following extraction in Reports 38 and 39 caused subacute bacterial endocarditis is improbable because the interval between the extraction and the onset of symptoms is hardly long enough. Again in Report 29 the interval was too long for extraction to be a probable cause of endocarditis. In Report 40 the interval was not indicated.

Observations other than case reports substantiate the idea that the bacteremia following dental extraction may cause subacute bacterial endocarditis. Isolation of the same organism from the apices of teeth and the blood or valvular vegetation of an individual with bacterial endocarditis has been reported by several. Blummer<sup>5</sup> stated that "it was possible to isolate the same organism (*Str. viridans*) from the apices of the teeth at the same time it was present in the blood." Beck<sup>55</sup> reported the same finding. The serologic similarity if not identity of the organism was not reported by Blummer, but was established by Beck. Burket,<sup>35</sup> culturing from teeth removed in situ at autopsy, mentions a case of subacute bacterial endocarditis in which the onset of symptoms was definitely related to the removal of an abscessed tooth. *Str. viridans* was found in the blood stream and at the periapex of a tooth during life, and in the viscera at autopsy. The organism found in the viscera was not serologically identical with the one found in the periapex of the extracted tooth. Lesbre and Granelaud<sup>60</sup> made the same observation as Beck, but instead of determining the serologic similarity of the two organisms, they established similarity between the organisms by animal inoculation. They stated that the same lesions were produced in rabbits by both organisms. O'Kell and Elliott<sup>38</sup> found serologic identity of streptococci cultured from the blood of patients with subacute bacterial endocarditis and the organism cultured from blood following extraction.

A paper reporting cured cases of subacute bacterial endocarditis<sup>46</sup> states that "two patients insisted that they were well until they underwent dental extraction." Northrop and Crowley<sup>44</sup> reviewed the records of 138 patients with subacute bacterial endocarditis and found that 23 gave histories of dental ex-

TABLE IV  
REPORTED CASES OF SUBACUTE BACTERIAL ENDOCARDITIS PROBABLY RESULTING FROM EXTRACTION OF TEETH

NO.	YEAR	AUTHOR	AGE	SEX	STATUS OF ENDOCARDITIS BEFORE EXTRACTION	INTERVAL	FIRST SYMPTOM OF SUBACUTE BACTERIAL ENDOCARDITIS	EVIDENCE OF SUBACUTE BACTERIAL ENDOCARDITIS	COMMENTS AND CONCLUSION
17	1913	Hemstead <sup>54</sup>	21	F	Malformation of right ventricle. No evidence of endocarditis	"The next day,"	Cold sensations. Pain in the back, followed by fever, profuse sweating, and septic fever	Culture of blood positive for <i>Str. viridans</i> . Recovery reported	Subacute bacterial endocarditis not verified. Postextraction bacteremia as cause obscured by delivery of baby
18	1932	Beck <sup>55</sup>	40	M	Showed some improvement from attack of active rheumatic endocarditis	Not stated. Root canals were cultured and found <i>Str. viridans</i> identical, bacteriologically and serologically, with those isolated from the blood	Not stated	Blood positive for <i>Str. viridans</i>	No conclusion because interval is not indicated. Good evidence that dental extraction caused subacute bacterial endocarditis on valve previously affected by rheumatic fever
19		Vanderhoof <sup>56</sup> Case 2	48	F	No evidence of chronic or active rheumatic endocarditis	Four or 5 weeks	Chill and fever	Petechiae. Osler's nodes. Blood positive for <i>Str. viridans</i>	Cause of subacute bacterial endocarditis uncertain because 4- or 5-week interval increases possibility of infection such as common cold occurring in interim and causing the endocarditis



20	Von Phul <sup>18</sup> Case 3	21 M	History of rheumatic fever in childhood. No evidence of rheumatic fever at time of extraction	Forty-three days	"Tired," short of breath, pains in abdomen. Culture of blood positive for <i>Str. viridans</i>	Large vegetation of mitral and aortic valves	Probably resulted from dental extraction. In-sidious symptoms may have existed sooner than 43 days after ex-traction. Interval of this length admits pos-sibility of common cold causing the endocardi-tis
21	1934 Weiss Case 2	30 F	Probably rheumatic fever at earlier date. Some evidence of ac-tive rheumatic fever or cardiac failure at time of extraction	Immediately	Malaise, chilly sensa-tions, fever followed by a chill, redness and tenderness of finger tips	Blood yielded <i>Str. viridans</i> . Café au lait complexion	Some evidence of symp-toms of endocarditis at time of extraction, mak-ing it impossible to de-termine if subacute bac-terial endocarditis was present before extrac-tion
22	Weiss Case 6	16 F	"Congenital heart le-sion." No evidence of endocarditis	Four weeks	Fatigue, malaise, blurring of vision	Culture of blood positive for <i>Str. viridans</i>	Proof of subacute bac-terial endocarditis is lacking; probably sub-acute bacterial endo-carditis resulting from dental extraction
23	Weiss Case 7	8 F	History of chorea. Child apparently well at time of ex-traction	Two weeks	General weakness, malaise, increased pallor, and reported paralysis of all ex-tremities for 3 days	Blood positive for <i>Str. viridans</i> . Neu-rological evidence of left cerebral thrombosis	Cannot establish state of activity of endocarditis before extraction. Can-not be sure that sub-acute bacterial endo-carditis was present, as only good evidence is blood positive for <i>Str. viridans</i>

TABLE IV—CONT'D

NO.	YEAR	AUTHOR	AGE	SEX	STATUS OF ENDOCARDITIS BEFORE EXTRACTION	INTERVAL	FIRST SYMPTOM OF SUBACUTE BACTERIAL ENDOCARDITIS	EVIDENCE OF SUBACUTE BACTERIAL ENDOCARDITIS	COMMENTS AND CONCLUSION
24	1938	Feldman, Trace <sup>51</sup> Case 1	36	F	Evidence (dyspnea) of cardiac failure. No evidence of acute rheumatic heart disease or subacute bacterial endocarditis	Immediately	Fever, palpitation, weakness, increased dyspnea	Blood positive for <i>Str. viridans</i> . Vegetation on mitral valve	Teeth were apparently removed in effort to relieve cardiac failure. Interval short for subacute bacterial endocarditis to result from extraction
25		Case 5	48	F	No evidence of having ever had rheumatic fever or any type of endocarditis. A possible bicuspid aortic valve suggested. No autopsy	No definite symptoms until "several weeks" later	Low-grade fever, increase in pulse rate. Anemia	Change in heart sounds, blood positive for <i>Str. viridans</i> only at end of disease	Interval between extraction and onset not definite. An intervening upper respiratory infection could have been responsible for subacute bacterial endocarditis. No conclusive evidence of subacute bacterial endocarditis
26	1939	Sale <sup>57</sup> Case 1	5	M	Chronic rheumatic endocarditis. Auricular fibrillation	About 1 week	Weak, fever, chills, Libman's nodules. Blood culture positive for <i>Str. viridans</i>	"Fibrous process upon which is superimposed a fibropurulent exudate." Bacterial clumps in vegetation of mitral valve	Not stated as being subacute bacterial endocarditis by author. Auricular fibrillation present at time of onset makes it doubtful if there was subacute bacterial endocarditis

traction shortly before the onset of subacute bacterial endocarditis. Whether a chronic endocarditis was present at the time of extraction was not stated by either Smith, Sauls, and Stone, or by Northrop and Crowley.

Hopkins<sup>58</sup> reported a mycotic aneurysm which resulted apparently from a postextraction bacteremia. In his fifth report he stated that a woman with hypertensive heart disease had several teeth removed. Severe occipital headache and dizziness followed. A week later a septic fever was present. A mycotic aneurysm was found at autopsy. There was no mention that any symptoms of endocarditis were present at the time of extraction.

In the textbook, *Osler's Principles and Practice of Medicine*,<sup>4</sup> Christian states that "patients with rheumatic or congenital heart disease are candidates for subacute bacterial endocarditis and if surgical manipulation (of the teeth) is needed, sulfanilamide should be given forty-eight hours before and after the procedure."

While most writers feel that subacute bacterial endocarditis is likely to occur on the valve affected by chronic endocarditis, Von Glahn and Pappenheimer feel that it is the valve with recent or active rheumatic vegetations which is most likely to be the site of subacute bacterial endocarditis. If more evidence were set forth in every report as to the condition of the valves before dental manipulation, the position of extraction with regard to valvular damage and subacute bacterial endocarditis might be determined more accurately.

Measures to prevent the subacute bacterial endocarditis resulting from extraction have been considered by those submitting case reports. Prophylactic efforts are suggested in three directions. The first step proposed is to obtain some information as to the cardiac condition before extraction. Von Phul,<sup>56</sup> Hopkins,<sup>58</sup> and Geiger<sup>53</sup> recognize the need of inquiry as to the condition of the patient's heart before an extraction is performed. Geiger points out that an abscessed tooth should not be extracted without some assurance that the patient is free from valvular disease. Sale emphasizes closer cooperation between the dentist and physician.

The second general step proposed to prevent endocarditis resulting from extraction is judgment and care in extraction. While there is general agreement that oral infection should be eliminated,<sup>2, 5, 51</sup> Geiger<sup>53</sup> rightfully opposes indiscriminate extraction, and points out that gingival infection should be cleared up before extraction.

Sterility of the dental instruments is thought by Bernstein<sup>49</sup> to be of value in preventing active endocarditis. Abrahamson,<sup>48</sup> having observed untoward effects apparently resulting from local dental anesthesia, believes that general anesthesia should be employed. In considering the importance of sterile instruments, it must be remembered that the tooth and contiguous structures rather than the instruments are the source of the *Str. viridans*. Conduction anesthesia has largely replaced the infiltration method, and Abrahamson's objection to local anesthesia can be overcome.

The prophylactic use of sulfanilamide compounds is the third proposed action for the prevention of endocarditis from postextraction bacteremia. Geiger<sup>53</sup> suggests that sulfadiazine might be used before extraction in the situation of a patient with cardiac disease. He states that "it is acknowledged that none of the sulfonamide drugs" are active against *Str. viridans*. Smith,

TABLE V  
REPORTED CASES OF SUBACUTE BACTERIAL ENDOCARDITIS WHICH MAY HAVE RESULTED FROM EXTRACTION OF TEETH

NO	YEAR	AUTHOR	AGE	SEX	STATUS OF ENDO-CARDITIS BEFORE EXTRACTION	INTERVAL	FIRST SYMPTOM OF SUBACUTE BACTERIAL ENDOCARDITIS	EVIDENCE OF SUB-ACUTE BACTERIAL ENDOCARDITIS	COMMENTS AND CONCLUSION
27	1931	Abrahamson <sup>48</sup> Case 2	53	M	Indefinite. Either acute rheumatic endocarditis or cardiac failure at time of extraction	About 1 month	Excessive bleeding followed extraction. Purpura hemorrhagica all over body within one month	No valid evidence. Blood cultures negative. No petechiae, Osler's nodes, or embolic phenomenon	Insufficient evidence of this was subacute bacterial endocarditis
28	1931	Abrahamson	53	M	Moderate enlargement. "Systolic murmur, maximal in mitral area." Repeated attacks of paroxysmal fibrillation. General deterioration of health and loss of weight at time of extraction	About 60 days after first extraction, and 3 days after second extraction	"Purpuric eruption," Hematuria. Paralysis of left arm	None except inclusion of this report in a series stated to be of subacute bacterial endocarditis	No stated evidence that this was subacute bacterial endocarditis
29	1934	Von Phulse <sup>49</sup> Case 1	30	F	Not clear	Five months. During this time patient was delivered of baby	Fever	Autopsy findings were those of subacute bacterial endocarditis	Because of interval and delivery, it seems improbable that dental extraction caused the endocarditis
30	1939	Hopkins <sup>55</sup> Case 4	60	M	Complained of "heart trouble." No accurate description of preexisting lesion	Interval after first extraction not stated. Following second multiple extraction, symptoms "immediately."	Following first extraction, chills and fever. After second extraction, chills, afternoon fever, lassitude, loss of strength, and night sweats	<i>Str. viridans</i> recovered from blood stream. "The course was septic." No autopsy	No further clues as to nature of "heart trouble." Not evident that subacute bacterial endocarditis is present
31	1939	Salz <sup>57</sup> Case 2	72	M	No definite history of valvular damage. History of frequent "fainting spells" upon exertion. No evidence of activity at time of extraction. Was receiving treatment for prostatic hypertrophy at time of extraction	Seemed to follow very soon after extraction	Swelling of jaw. Slight fever followed by chills and increased fever	No complete autopsy. Thickening of mitral valve with "engrafted lesions the result of a recent endocarditis." No bacteriologic findings reported. Duration of disease 5 months	No conclusive evidence of subacute bacterial endocarditis. Treatment for benign hypertrophy of prostate could have been cause. Dental extraction may have caused subacute bacterial endocarditis in this man



32	1939	Elliott <sup>41</sup>	28	M	"Rheumatic carditis"	During dental treatment	Not stated	Not stated	Reports by Elliott cannot be properly evaluated because evidence of state of endocarditis too indefinite and no proof that patient had subacute bacterial endocarditis
33		Case 1	40	M	"Rheumatic carditis"	Two weeks	Not stated	Not stated	
34		Case 3	19	F	Patent ductus arteriosus	Ten days	Not stated	Not stated	
35		Case 4	25	F	Indefinite	Less than 9 weeks	Not stated	Not stated	
36		Case 5	34	M	Indefinite	Two days	Not stated	Not stated	
37		Case 6	59	M		Three weeks	Not stated	Not stated	
38	1939	Hopkins <sup>58</sup>	44	M	Congenital cardiac lesion as confirmed by autopsy. No evidence of active endocarditis at time of extraction.	"Immediately"	Weakness, sweating, mild prostration. Septic type of fever	<i>Str. viridans</i> from blood and pericardial fluid. Vegetation on pulmonary valve leaflets. Mycotic aneurysm of pulmonary artery	Interval was short to admit extraction as cause of subacute bacterial endocarditis
39		Case 2	35	F	History of rheumatic fever at 4 years of age. No evidence of rheumatic activity at time of extraction	No extraction. Dental treatment for pyorrhea employing novocain. Symptoms within 12 hours	Chills, fever, sweats	Vegetation on mitral valve	The 12-hour interval was short to admit dental treatment as cause of subacute bacterial endocarditis
40	1941	Paquin <sup>59</sup>	38	M	Mitral valve insufficiency. Evidence at autopsy of chronic rheumatic endocarditis	"Thereafter." Less than 5 months	Pain in both legs. Influenza, weak, tired, chills, muscular pains	Cultures of blood yielded <i>Str. viridans</i> . Subacute bacterial endocarditis of mitral valve at autopsy	Conclusions as to relation between extraction and endocarditis cannot be drawn because: (1) Interval is indefinite. (2) Patient had influenza after extraction. (3) First symptoms following extraction suggest cardiac failure

Sauls, and Stone<sup>40</sup> as well as Dick<sup>41</sup> report cures of subacute bacterial endocarditis using sulfadiazine, but this favorable therapeutic result has not been the usual clinical experience. Budnitz, Nizel, and Berg<sup>52</sup> administered sulfapyridine before extraction to 27 patients with valvular disease or congenital anomalies of the heart. The culture of blood obtained after extraction from the 27 patients was uniformly negative. No untoward results followed extraction in any of the patients. Hopkins is stated by Northrop and Crowley<sup>44</sup> to have found (unpublished data) sterile blood in an unstated number of patients following extraction when premedication with sulfonic drugs was used.

Northrop and Crowley, working with 73 patients, first took preoperative cultures, all of which were negative. A sulfathiazole level of less than 3 mg. per 100 c.c. of blood was obtained in 50 patients. Following dental surgery, *Str. viridans* was isolated three times in the first group of 23, making 17 per cent of cultures positive for *Str. viridans*. In the group of 50 with a blood level of 3 to 5.6 mg. per 100 c.c., *Str. viridans* was isolated once.

Northrop and Crowley's control group consisted of 93 patients of random sampling, who received no premedication. There were six postextraction cultures which were positive for *Str. viridans*. There was, then, a decided reduction in the incidence of bacteremias in the group of 50 patients in which a level of sulfathiazole of over 5 mg. per cent was reached.

Schwartz and Salman<sup>62</sup> expressed an opinion contrary to that of those writers who are in agreement that extraction may cause subacute bacterial endocarditis in the case of an individual with previous valvular damage. The two writers found that extraction under local anesthesia of 467 teeth in 136 children in both the active and inactive phase of rheumatic fever was followed by death only once. Their conclusion was that the extraction of teeth in patients with rheumatic fever entails no greater risk than in any other group.

In a critical analysis of Schwartz and Salman's observations, it should be pointed out that Blummer, Thayer, Clawson and Bell and others have included studies of the ages at which people are attacked by subacute bacterial endocarditis. The incidence of the disease in people under 14 years is rather low, perhaps 12 per cent. As Schwartz and Salman point out, Christian states<sup>9</sup> that subacute bacterial endocarditis does not occur in individuals with auricular fibrillation, and that the absence of this rhythm is a point in the determinative background of subacute bacterial endocarditis. An examination of the data presented by Schwartz and Salman reveals that, of the 136 patients, only 38 were likely to be attacked by subacute bacterial endocarditis, the others being under 14 years of age or having auricular fibrillation at the time of extraction.

Dental infection may result from, as well as cause, a systemic infection. In 1931 Rickert<sup>63</sup> recognized that hematogenous infection might secondarily involve the apices of teeth. Recent work has demonstrated a mechanism by which incipient subacute bacterial endocarditis may, by producing a pulpitis, be responsible for the extraction of teeth. Robinson and Boling<sup>64</sup> showed that the traumatized dental pulp in a considerable number of cases is a site of predilection for inflammation resulting from bacteria in the blood stream. Extraction of a tooth, because of anachoretic pulpitis of endocardial origin, would be followed by signs of endocarditis because of the clinical course of subacute bacterial endocarditis. The patient, turning naturally to some incident, relates

the onset of symptoms of the endocarditis to the extraction of the tooth. There is not at present enough evidence to prove whether or not anachoretic pulpitis of endocardial origin is a reason for the removal of teeth. A series of cases of the symptoms of subacute bacterial endocarditis following shortly after extraction of teeth and in which the dental history had been written in detail would be of great help in investigating this problem. In such a history, the specific reasons for extracting the teeth would be brought out. The particular reasons for extraction in the cases reported in the literature as well as in the case studies described in the next section are not stated often enough to draw conclusions as to the state of the dental pulp.

#### MATERIALS AND METHODS

The hospital records for study were obtained by an examination of the autopsy protocols of the Department of Pathology of the Washington University School of Medicine. These protocols included all autopsies performed by the Department of Pathology through the year 1942. The first protocol was dated 1910, and 10,300 were recorded in all during the 32-year span.

The 10,300 protocols were examined to select all cases of subacute bacterial endocarditis which had come to autopsy. The hospital records of those patients were then read in order to determine whether or not any dental procedure seemed to bear a significant relation to the systemic disease. Those cases in which the relation between the dental procedure and the endocarditis seemed to be of some significance were included in the study.

Of necessity, certain definitions of terminology had to be made in the course of this procedure. Although subacute bacterial endocarditis has been studied exhaustively, there is disagreement as to its exact definition. It is particularly difficult to separate subacute bacterial endocarditis from either acute endocarditis or acute rheumatic endocarditis. Any such separation necessarily must be arbitrary. For the purposes of the study, the following criteria for the determination of subacute bacterial endocarditis were set up, and only those cases which met these criteria were included in the study.

1. The onset of the disease must have been insidious.
2. The patient must have lived at least two months after the appearance of symptoms recognizable as those of subacute bacterial endocarditis.
3. The morphologic changes of the heart found in subacute bacterial endocarditis must have taken place. The important consideration here was that fairly firm, moderately friable vegetations of between 1 mm. and 1 cm. in diameter must be present.
4. In addition to the above, one of the following requirements must have been met: (a) A positive culture of blood must have been found, or (b) embolic phenomena must have been found.

1. *Description of Cases Selected for the Study.*—Subacute bacterial endocarditis was found in 92 of the 10,300 consecutive protocols examined. When the hospital records were read, an apparent sequential relation between subacute bacterial endocarditis and extraction was found in eight of the 92 cases. No dental procedure other than extraction was mentioned.

Upon closer examination of these eight cases it was felt that in two the relation of the extraction to the disease was not clear-cut enough to permit the

inclusion of the case in the significant group. In the first of these instances the extraction was shortly followed by symptoms of subacute bacterial endocarditis and it may be that a causal relation existed. The history prior to extraction was not given in enough detail in the hospital record, however, to exclude the possibility that the patient had some symptoms of subacute bacterial endocarditis before the extraction. Because of this lack of evidence the case was not included.

In the second of the two cases which were excluded from the significant group the patient was known to have had subacute bacterial endocarditis prior to the extraction. The relation in this case was complicated by syphilis and the fact that the teeth were abscessed. It was therefore decided not to include the case.

The six cases remaining for study are presented.

2. *Abstracted Reports of the Selected Cases.*—CASE A.—Autopsy No. 6015. A 26-year-old white woman had had rheumatic heart disease at the age of 14 years. The patient was well until December, 1934, when a tooth was removed without immediate untoward effects. About five days later the patient "caught cold" and this was followed immediately by malaise, chills, and fever. The patient was prostrate on January 24, and on February 1 a culture of the blood yielded *Str. viridans*. The patient died the same day.

The pertinent findings at autopsy were chronic endocarditis of the mitral valve, subacute bacterial endocarditis (*Str. viridans*) of the mitral valve and left auricular wall, hypertrophy and dilatation of the heart, moderate focal fibrosis of the myocardium, abscesses of the right lower lobe of the lung, and an infarct of the right lower lobe.

*Discussion.*—In view of the fact that subacute bacterial endocarditis may follow an upper respiratory infection (Weiss, Blummer), it is impossible to say whether the upper respiratory infection or the extraction of the tooth caused the subacute bacterial endocarditis. There is no evidence that the rheumatic endocarditis was active at the time the tooth was extracted.

CASE B.—Autopsy No. 6238. A 33-year-old woman had chorea at 12 years of age and was told she had enlargement of the heart. She went through a normal pregnancy in January, 1935. Following delivery she did not regain strength, and this was considered the onset of the terminal cardiac disease. In February she noticed "sore spots at the ends of her fingers," malaise, and weakness. In June, sixteen teeth of the upper jaw were extracted because of radiographic evidence of periapical abscesses. Chills, fever, malaise, and sweating were complained of in the following days. The patient's physician reported culturing the "pneumonia germ" from her blood and found her anemic. Two months later she developed a migratory polyarthritides. *Str. viridans* was cultured from the blood on one occasion. The patient died Sept. 9, 1935, nine months after the onset of the disease.

The pertinent findings at autopsy were chronic endocarditis of the mitral valve, subacute bacterial endocarditis (*Str. viridans*), infarcts of the spleen with abscess formation, infarcts of the kidneys and brain, mycotic aneurysm of the splenic artery, chronic nephritis, hypertrophy and dilatation of the heart, and focal fibrosis of the myocardium.



*Discussion.*—This patient had rheumatic fever at the age of 12 years. It was not active until after she delivered a baby, when Osler's nodes, malaise, and weakness were present. The patient explained the sore finger tips on the basis of the kind of work she was doing. This is to be discredited because it does not explain the weakness and malaise. These symptoms mark the onset of subacute bacterial endocarditis. Six months later, sixteen teeth were extracted probably hoping to relieve symptoms actually those of endocarditis. The extraction was followed by chills, fever, sweating, and malaise. The extraction may have accelerated the course of the subacute bacterial endocarditis.

CASE C.—Autopsy No. 8775. This 32-year-old man had been told that he had "a bad heart." For the past two years he had malaise and felt "tired out" without apparent reason. In March, 1940, he had four teeth extracted for the purpose of relieving this "tired feeling." The record states that "since the extraction of the teeth" he had fever, chills, and pains in the legs, arms, and toes. It was not possible to determine the exact interim between the extraction of the teeth and these symptoms. On July 23, 1940, a culture of blood was positive for *Str. viridans* and there were petechiae present. The patient died on July 25, 1940.

The pertinent findings at autopsy were chronic endocarditis of a malformed aortic valve, hypertrophy and dilatation of the heart, subacute bacterial endocarditis of the mitral and aortic valves, petechiae of the skin and conjunctiva, infarcts of the spleen and kidney, and focal embolic glomerulonephritis. Microscopic examination of the myocardium showed lesions compatible with healed Aschoff bodies.

*Discussion.*—The important question as to whether the patient had active rheumatic endocarditis or cardiac failure at the time the teeth were extracted cannot be answered from the information obtained from the record. As there were no other incidents to explain the onset of subacute bacterial endocarditis, it is concluded that the disease resulted from a bacteremia following the extraction of teeth.

CASE D.—Autopsy No. 9325. A 55-year-old woman had been told at the age of 40 that she had a heart murmur. Since the summer of 1939, two years before her death she had been confined to bed about half the time because of weakness. Since January, 1940, she had complained of increased weakness, questionable chills, belching, an uneasy feeling around the heart, and a loss of 20 pounds. Since May 1, 1940, she had night sweats each night. In February, 1940, eight teeth were extracted, and this was followed by excessive bleeding. The first blood culture was taken May 21 and was positive for *Str. viridans*. Following this were fifteen positive and eleven negative cultures of blood. Petechiae were visible and the spleen became palpable. The patient died on July 24, 1941.

The pertinent findings at autopsy were subacute bacterial endocarditis of the mitral valve (*Str. viridans*), chronic endocarditis of the mitral valve, hemorrhage into the right frontal lobe of the brain with rupture into the ventricle, healed infarcts of the left kidney, and petechiae of the conjunctiva of the left eye.

*Discussion.*—Here is a patient with cardiac failure but with no evidence of endocarditis from the summer of 1939 to May, 1940. The extraction of the teeth occurred eight weeks before the night sweats, which were the first sign of endocarditis. The extraction may have been incidental to the cardiac failure. The removal of sixteen teeth suggests an effort to relieve a general weakness and loss of health by removing oral foci of infection. Since the cardiac failure was becoming more marked before the teeth were extracted, as evidenced by the increased weakness and loss of weight, the extraction probably had no effect on the course of the cardiac failure. Unless one is to consider the subacute bacterial endocarditis as remaining subclinical for eight weeks, neither can the extraction be held as the cause of the endocarditis.

CASE E.—Autopsy No. 9539. An 18-year-old girl had scarlet fever as a child, and, at the age of 16, rheumatic fever from which she recovered without cardiac impairment. About Aug. 28, 1941, one of several carious teeth was extracted. Two weeks after extraction there was persistent elevation of the temperature, transitory paralysis of one extremity, migratory polyarthritides, subcutaneous nodules of the scalp, Osler's nodes, and petechiae. There is a contradiction in the record as to whether these symptoms were preceded by a sore throat. If there was a sore throat, it was immediately preceding the first of these symptoms. On Nov. 12, 1941, the first of five blood cultures yielding *Str. viridans* was taken. Embolic phenomena followed which included petechiae, hemiplegia, and splinter hemorrhage. The patient died on Nov. 28, 1941.

The pertinent findings at autopsy were acute rheumatic myocarditis, chronic endocarditis of the mitral and aortic valves, fibrous obliteration of the pericardial cavity, fibrous thickening of the endocardium of the left auricle, hypertrophy and dilatation of the heart, subacute bacterial endocarditis of the mitral and aortic valves, rupture of the superior mesenteric artery with massive hemorrhage into the mesentery, hemorrhage into the left internal capsule of the brain, subarachnoidal hemorrhage over the left temporal and parietal lobes and base of the brain and over the right temporal lobe, healed infarcts of the spleen and right kidney, and petechiae of the conjunctiva of the eyes.

*Discussion.*—There were no signs of active endocarditis when the carious tooth was extracted. The symptoms of active rheumatic endocarditis, i.e., subcutaneous nodules, persistent temperature, and migratory polyarthritides, were followed by those of subacute bacterial endocarditis, Osler's nodes, petechiae, and a positive culture of blood. It is recognized that an upper respiratory infection may precede active rheumatic endocarditis, but if it is a causative factor it precedes the first symptoms of active endocarditis from seven to twenty-one days (Wilson). Assuming that the patient had a sore throat, it could not be considered a cause of the active rheumatic endocarditis because it occurred immediately before the first symptoms. The sore throat could be responsible for the subacute bacterial endocarditis, as it may have been in Case A. The extraction of the tooth preceded the onset of the symptoms of active rheumatic endocarditis by two weeks, and in the same way that a sore throat may cause active rheumatic endocarditis, the extraction probably caused the chronic endocarditis to become active.

CASE F.—Autopsy No. 9944. A 24-year-old woman had rheumatic fever at 8 and at 12 years of age. At 22 years of age, in May, 1941, the patient had lobar pneumonia at the time of the delivery of a baby. The pneumonia was followed by a cough which was productive of mucoid material and persisted for five months. During this time she had a chilly sensation, night sweats, and a loss of 6 pounds in four months. The record indicates that there was malaise in July, 1941, when an abscessed tooth was extracted. The extraction was followed by a "dry socket" and a pledget of cotton was removed from the dental alveolus three weeks after extraction. This was followed after two or three days by chills and fever four times a week, followed each time by sore finger tips and petechiae. At this time the patient had pains in the joints of the hips to the extent of being unable to walk. All blood cultures taken during a hospital admission of Sept. 14, 1941, were negative although a diagnosis of "probable subacute bacterial endocarditis" was made. During this hospital admission, radiographic evidence suggestive of bronchiectasis was found. On Dec. 29, 1941, the patient's blood was positive for *Str. viridans*. During the spring and summer of 1942, nine cultures of blood were positive for *Str. viridans*. Death was on July 26, 1942.

The pertinent findings at autopsy were chronic endocarditis of the mitral and tricuspid valves, hypertrophy and dilatation of the heart, subacute bacterial endocarditis of the left auricle, mitral valve, and left ventricle, petechiae of the skin, focal embolic glomerulonephritis, and aneurysm of a vein of the brain.

*Discussion.*—The symptoms of a productive cough, chilly sensations, night sweats, and loss of weight following delivery and lobar pneumonia were on the basis of pulmonary inflammation and the usual post-partum course of patients with marked cardiac impairment. There were no signs of subacute bacterial endocarditis until appearance of the "dry socket." The fever, chills, and petechiae mark the onset of the subacute bacterial endocarditis. There is no strong evidence of active rheumatic endocarditis at the time of extraction of the teeth.

A study of these six cases of subacute bacterial endocarditis suggests four positions that extraction may bear to cardiac disease:

1. Extraction may cause subacute bacterial endocarditis (Cases C, F, and perhaps A).
2. Extraction may be performed after the onset of endocarditis lenta but appear to have caused the infection (Case B).
3. Extraction may cause chronic rheumatic endocarditis to become active (Case E).
4. Extraction may be performed during cardiac failure, probably in an effort to relieve the symptoms of cardiac failure (Case D). (Extraction in this situation has no definite effect on cardiac failure or endocarditis.)

#### DISCUSSION

1. *The Bilateral Nature of the Relation Between Dental Procedures and Endocarditis Lenta.*—There is a good deal of evidence from the reports in the literature and from the cases presented that an association exists between cardiac disease and extraction. Extraction sometimes seems to be performed to relieve



symptoms actually those of endocarditis. On the other hand, endocarditis sometimes follows extraction in such a way as to suggest a causal relation. On further inquiry into cases of the latter kind, it appears that in some there may be a true causal relation, but that in others the endocarditis was actually present before the extraction took place but was unrecognized and because of the sequence of events it has been attributed to the extraction.

2. *Extraction as a Cause of Subacute Bacterial Endocarditis.*—A considerable amount of evidence has been presented that the extraction of teeth sometimes causes subacute bacterial endocarditis. Indication of the causal relation of extraction to subacute bacterial endocarditis is of three types.

The first evidence is the finding of *Str. viridans* at the apex of the tooth and in the blood during the endocarditis. Blummer<sup>5</sup> and Thayer<sup>2</sup> point out that *Str. viridans* is nearly always the cause of endocarditis lenta. It has been pointed out also that this organism is the one most frequently found in and around the apices of teeth. More specific evidence that it is the organism which, when liberated into the blood, causes infective endocarditis was presented by O'Kell and Elliott.<sup>38</sup> While the organism found in the apices of teeth and concurrently in the blood stream was not serologically identical in the case studied by Burket, this identity was established by Beck. Lesbre and Granclaude reported inferential evidence that the *Str. viridans* found in the blood stream was identical with the organism found at the same time at the apex of an extracted tooth during subacute bacterial endocarditis.

It is believed that the *Str. viridans* found in the apical region of teeth causes endocarditis in some cases. Reports of cultures from apices should be regarded critically, however, because the organisms cultured from the apices of extracted teeth represent those of the gingival flora as well as of the root apex.

The second kind of evidence of the causal relation between extraction and endocarditis consists in the descriptions of the streptococcal bacteremia which sometimes follows the extraction of teeth. While the wide variation between percentages of positive cultures reported by the different workers indicates that the factors which determine the likelihood of postextraction bacteremia are not yet understood, there is proof that a streptococcal bacteremia following extraction often occurs. From the review of the literature presented in Table I it appears that about 20 per cent of all extractions result in a bacteremia which includes, among other organisms, *Str. viridans*. The bacteremia is the reasonable explanation of the pathogenesis of a subacute bacterial endocarditis following dental manipulation.

Perhaps the third type of evidence is the most direct and convincing. A critically analyzed series of reports found in the literature makes it evident that subacute bacterial endocarditis follows extractions and other dental manipulations. Forty reports of subacute bacterial endocarditis supposedly resulting from the extraction of teeth were found. After critical analysis, fifteen of these were found to bear every evidence that the endocarditis did result from extraction, and in ten others the relation seemed probable. Cases C and F of this study may be added to this first group, and Case A to the second, making a total of twenty-eight cases in which extraction probably caused subacute bacterial endocarditis.



While actual proof that extraction sometimes causes subacute bacterial endocarditis is lacking, the strength of the evidence set forth justifies a belief that this relation between dental extraction and subacute bacterial endocarditis rests on the evidence presented that extraction sometimes causes this form of endocarditis.

While no estimate of the percentage of cases of subacute bacterial endocarditis resulting from dental extraction was found, if conclusions can be drawn from the cases collected from 10,300 autopsies, dental manipulations cause 2 or 3 per cent of the subacute bacterial endocarditis.

It is stated (Christian, Blummer, Levine and Fulton) that males are attacked by subacute bacterial endocarditis more frequently than females. A consideration of incidence as to sex, based on twenty-five collected reports and three studied cases of subacute bacterial endocarditis probably resulting from the extraction of teeth, may be of interest.

TABLE VI  
SEX OF PATIENTS WITH ENDOCARDITIS RESULTING PROBABLY FROM THE EXTRACTION  
OF TEETH

MALES		FEMALES	
NUMBER	PER CENT	NUMBER	PER CENT
13	46.4	15	53.5

As can be seen from Table VI, there is no significant correlation between sex and the incidence of subacute bacterial endocarditis resulting from the extraction of teeth.

Knowledge that a certain disease usually attacks individuals within a given range of ages is of practical importance in preventing that disease. Blummer<sup>5</sup> (page 120) found that 56 per cent of the 317 patients in his series were between 20 and 40 years of age. With the exception of Reports 3 and 6 in Table II the range of ages in the other thirteen was from 18 to 39 years. Including Reports 3 and 6 the average age was 27.3 years. Ages in the three studied cases in which endocarditis seemed to result from extraction were 24, 26, and 32 years, and hence within the range of the collected reports. It is concluded that the age of patients with subacute bacterial endocarditis resulting from dental extraction does not differ greatly from the age of patients with subacute bacterial endocarditis resulting from other causes.

Several persons have emphasized that endocarditis lenta usually occurs on a previously damaged or malformed valve and in the absence of auricular fibrillation. Endocarditis resulting from extraction is not an exception to this general observation. Of the fifteen reports reviewed in Table II, only Report 2 had no history of previous valvular disease. Damage by congenital malformation was found only once, in Case F of the cases studied. In the collected reports, there was a congenital heart lesion in only one, Report 22. Proof of endocarditis lenta is lacking in this case. Auricular fibrillation was not noted in any of the reports.

It is difficult to establish whether a valve with the chronic or acute form of rheumatic endocarditis is more susceptible to the bacteremia which sometimes follows dental extraction. Either very specific clinical findings at the time of extraction or Aschoff bodies in the myocardium must be found to justify the conclusion that rheumatic activity was present at the time of extraction. Of the

fifteen reports in Table II there is evidence that fresh vegetations of active rheumatic endocarditis may have been present at the time of extraction in three reports (1, 8, and 10). It is stated in Report 1 that the patient had recently recovered from active rheumatic fever, and in Report 10 it is stated the patient had active rheumatic fever at the time of the extraction. In Report 8 it is stated that the teeth were removed to relieve arthralgia. In Case C of the present series, microscopic lesions compatible with healed Aschoff bodies were seen. Studies indicate that subacute bacterial endocarditis is more likely to occur on valves with active vegetations than on valves with chronic vegetations and from this fact it is deduced that extraction during the active phase of rheumatic endocarditis is particularly hazardous to the patient with pancarditis.

The initial signs and symptoms of endocarditis lenta may be easily overlooked, as the infection has an insidious onset. These signs were chills, fever, and malaise in both the collected reports and studied cases. Following these symptoms of early subacute bacterial endocarditis there was anemia, growth in cultured blood, and often embolic phenomena. Other early symptoms mentioned in the reported cases were the feeling of being depressed, cold sensations, and palpitation. Excessive bleeding following extraction was noted in three of the reports (2, 5, and 27). Bleeding was noticed only in Case D of those studied for this paper, and in this case it was probably on the basis of the extraction of many teeth within a short period of time. The bleeding in the reported cases could be explained easily if there were fibrinolytic streptococci present around the teeth at the time of extraction.

Since subacute bacterial endocarditis is initiated by the establishment of an organism on a heart valve, it would seem that the interval between the entry of the organism and the first clinical symptoms would be fairly constant. If the streptococci enter the blood stream by the extraction of a tooth, then it would seem that the interval between dental manipulation and the onset of symptoms would be fairly uniform. The interim is probably relatively constant, but because the exact date of the extraction is not often remembered, and the onset of the disease is insidious, it seems to vary. The first manifestations of subacute bacterial endocarditis are not always remembered, adding to the difficulty of determining the interval between the extraction and onset of the disease.

The first symptoms of subacute bacterial endocarditis supposedly result from the bacteremia and from the presence of vegetations on the valves. If endocarditis lenta results from a postextraction bacteremia, the interval between the extraction and onset of subacute bacterial endocarditis must be the period of formation of the vegetation. The apparent interval between extraction and the first sign of endocarditis in the cases studied was a few days in Case C and three weeks in Case F. The interval given in the reports is from three days to three weeks, approximately. In several reports the interval was described as "shortly," "a few days," and, in Report 1, not stated.

A period of three days to three weeks is compatible with the formation of a vegetation on the valve. A short interval, however, of a few hours to a day, between the extraction of teeth and the onset of symptoms is reason to doubt that the endocarditis resulted from the extraction. In reports in which the interval is less than the time required for the formation of a vegetation, the

malaise and fever may have resulted from an endocarditis present at the time of extraction or may have been caused by the postextraction bacteremia.

It is difficult to determine whether the malaise, chills, and fever which immediately follow extraction are caused by a postextraction bacteremia or whether these symptoms are those of incipient endocarditis. Report 7 states that there was great difficulty in removing the tooth. The chills and fever following this extraction were probably caused by the surgical trauma. In Reports 11, 17, 21, 24, 32, 37, and 38, the symptoms of malaise, fever, and chills were probably caused by subacute bacterial endocarditis present at the time of extraction.

Because of its insidious onset there seems little doubt that endocarditis lenta is sometimes present when the teeth are removed. It is natural for people to associate symptoms with some remembered incident. If a tooth were extracted about the time of the onset of manifestations of endocarditis, any symptoms present very likely would be attributed to the extraction. In Case B of the studied series, subacute bacterial endocarditis was present at the time of extraction as evidenced by Osler's nodes. No doubt some of the infections reported to have been caused by dental manipulation were present at the time of extraction. A more detailed history, taken at the time of extraction, probably would reveal that the patient had endocarditis lenta and perhaps that teeth were about to be extracted merely hoping to relieve symptoms of his infection.

Since fragments of thrombi containing *Str. viridans* from the loose valvular vegetations are common in subacute bacterial endocarditis, it is reasonable to think that streptococci from the thrombotic vegetation of a heart valve sometimes produce an anachoretic type of pulpitis. Robinson and Boling demonstrated experimentally the attraction of an irritated dental pulp for blood-borne bacteria. As lodgment of minute infected emboli in the capillary tuft of the glomeruli is a common finding, septic emboli in the dental pulp during subacute bacterial endocarditis may prove to be at least an occasional feature. Evidence that subacute bacterial endocarditis causes anachoretic pulpitis must await three kinds of study.

First, complete and detailed dental records must be kept as an integral part of hospital records. These dental records would be kept by dentists and would include notation of, and the reason for, recent extractions as well as other dental procedures. Such records could be compared with the medical records. A sequential relation between the extraction and onset of endocarditis might be established in this way.

Second, examination of pulps of a series of teeth extracted during endocarditis would add to the evidence that emboli lodge in the pulps of teeth. Sections of pulps would show abscesses resulting from thrombi.

Third, experimental subacute bacterial endocarditis would be of value, not only in confirming that pulpitis results from endocarditis lenta, but in clearly establishing the position of the removal of teeth to the endocarditis. The possibility of experimental pathology in this problem will be discussed at the close of the discussion. Endocarditis resembling human subacute bacterial endocarditis has been produced in dogs<sup>66</sup> and in rabbits.<sup>67</sup>

3. *The Recrudescence of Chronic Endocarditis by Postextraction Bacteremia.*—A third relation between extraction and cardiac disease is suggested when chronic rheumatic fever becomes active, following extraction. While the



cause of rheumatic endocarditis is not known, there is good evidence to believe that it is the response of hypersensitive tissue to an antigen.<sup>21</sup> The implication of postextraction bacteremia as a portal of entry for antigen in the form of bacteria is obvious. In Case E of the present series, chronic rheumatic endocarditis seemed to be exacerbated by extraction. In this report, the patient had no signs of active rheumatic endocarditis at the time of extraction. Two weeks following extraction there was migratory polyarthritides, transitory paralysis of one extremity, and subcutaneous nodules of the scalp. Following these symptoms of acute rheumatic endocarditis, there were symptoms of subacute bacterial endocarditis. If a pharyngitis occurred, which is not clearly stated in the record, it could not have been responsible for these symptoms of acute rheumatic endocarditis, for the sore throat, if present, immediately preceded the onset of symptoms. Wilson, in her book *Rheumatic Fever*,<sup>65</sup> states that active rheumatic fever resulting from an upper respiratory infection appears from one to three weeks after the onset of the respiratory infection. This necessary interval between the infection and onset of rheumatic fever precludes the pharyngitis as the cause of active rheumatic fever.

In this study of the relation between extraction and subacute bacterial endocarditis, diagnoses of active rheumatic fever were not collected from the protocols. Case E was found only because a diagnosis of subacute bacterial endocarditis was also made. A study paralleling the present one, of the relation of extraction to rheumatic fever, should be carried out to provide evidence that the bacteremia following dental manipulation sometimes causes chronic rheumatic fever to become active.

In one report, No. 16, of the cases collected from the literature, there was some indication that extraction caused chronic rheumatic endocarditis to become active. The evidence that an exacerbation of rheumatic endocarditis was caused by the extraction of teeth is that the patient had no symptoms of active rheumatic endocarditis at the time of extraction; one month after the extraction, pains in the limbs appeared. A diagnosis of subacute bacterial endocarditis was made, but no autopsy was performed.

4. *Extraction and Symptoms of Cardiac Failure.*—Three positions of extraction to cardiac disease have been discussed. A fourth appears to be that in which extraction is performed during cardiac failure, probably in an effort to relieve some of the symptoms of such failure. It is a matter of observation that extraction is performed to relieve weakness, loss of weight, gastrointestinal disturbances. These symptoms may be actually those of cardiac failure. The reason for the extraction of teeth in Case D was not stated. The extraction of eight teeth within a month's time suggests an effort to relieve the loss of weight, weakness, and gastrointestinal disturbance by removing what was thought to be a foci of infection. Collected Reports 21, 23, and 27 have some evidence that cardiac failure was present at the time of extraction.

It is often true that an initial report of an association of diseases brings attention to the relation, and in this way is productive of an increasingly larger number of reports of the same relation of diseases. Of the relations that exist between the extraction of teeth and cardiac disease, that dental manipulation with subsequent bacteremia in individuals with valvular damage sometimes causes



subacute bacterial endocarditis is the most convincing. If more attention were directed to a study of the relation between extraction and cardiac failure, and to the hypothesis that subacute bacterial endocarditis may be responsible for extraction, perhaps clarification of the latter relations would result.

5. *Inferences as to the Dental Management of Cardiac Patients.*—A study of the relations between extraction and cardiac disease not only brings out a causal association but also suggests possible measures which might be adopted to prevent such results. Such practices should also result in a further clarification of the relations between dental procedures and cardiac disease.

The question arises as to whether or not subacute bacterial endocarditis resulting from the removal of teeth would have occurred had the teeth not been extracted. Possibly retention of the teeth would have prevented endocarditis in the cases reported. Murry and Moosnick, as well as Elliott, have shown that the movement that teeth undergo during mastication produces a bacteremia in a high percentage of cases. The presence of pyorrhetic or infected teeth in an individual with valvular damage is equally dangerous, if not more so, than the extraction of the teeth. At present it seems that measures have been found which will, to a great extent, prevent the postextraction bacteremia. If this is possible, the removal of teeth for patients with valvular damage should be done more freely than in the situation of a comparable group with no valvular damage.

Prevention of the infective endocarditis resulting from dental extraction can be effected by determining whether the heart valves are susceptible to endocarditis lenta, and in such cases preventing a streptococcal bacteremia. An examination by a physician of every patient needing an extraction is obviously impractical. A history taken by the dentist will reveal in most cases whether a damaged valve is present.

There is evidence in the reports of Geiger, of Budnitz, Nizel, and Berg, and more specifically of Northrop and Crowley, that premedication by sulfonic drugs will reduce materially the probability of a postextraction streptococcal bacteremia.

Pre-extraction medication with sulfonic compounds of patients having no valvular damage or other definite indication for sulfonic prophylaxis is inadvisable. It is a general principle of therapeutics that a drug should not be given without a reason. As shown by Northrop and Crowley, blood levels of less than 3 mg. per cent of sulfathiazole are inefficient. The amount of this drug required to obtain a protecting blood level will produce nausea, dizziness, and cyanosis in some individuals. The decision as to when to administer a sulfonic compound must rest on the information obtained from the revealing questions asked of the patient by the dentist before extraction.

Any measures which will reduce the incidence and severity of postextraction bacteremias are worthy of clinical consideration.

Attention is first directed to the bearing of periodontal sepsis on bacteremia following extraction. Again, information as to what dental conditions are likely to result in a bacteremia following extraction would be valuable in preventing endocarditis lenta as an occasional sequela of extraction. The condition of the teeth and their surrounding structures was not adequately stated in either the case studies or collected reports.

Efforts directed at reducing periodontal infection should lower the incidence of bacteremia. Some indication contrary to the generalization that extraction of pyorrhetic teeth is more likely to result in bacteremia than the extraction of teeth in normal tissue is found in Elliott's work.<sup>41</sup> Extraction on twenty-one patients with no detectable pyorrhea was followed by streptococcal bacteremia eighteen times. Extraction on the same number of patients with marked pyorrhea resulted in bacteremia in only five instances.

Efforts to sterilize the gingival crevice before extraction probably reduce the incidence of postextraction bacteremia. The effect on the incidence of postextraction bacteremia of painting the gingival crevice with iodine is not conclusive.<sup>31</sup> Faillo<sup>43</sup> found that the apices of extracted teeth were more often sterile if the gingival crevice had been cauterized before extraction. An investigation carried out by Austin and Cook<sup>68</sup> indicates that sterilizing the gingival crevice before extraction prevents bacteria from entering the periapical area.

Aside from efforts to eliminate and sterilize the gingival crevice before extraction, the techniques of dental anesthesia and extraction have a bearing on the likelihood of a postextraction bacteremia. A study of the incidence of bacteremia following extraction as summarized in Table I indicates that higher incidence of bacteremia follows extraction under general than under local anesthesia. No evidence concerning the relation between the technique of extraction, the type of anesthesia used, and the probability of a resulting subacute bacterial endocarditis was found; only a few reports mentioned whether a general or local anesthetic was used. Abrahamson<sup>48</sup> stated that general anesthesia is the one choice in most patients with endocarditis because the injection of a solution under pressure into infected tissue disseminated bacteria. As a matter of clinical observation, more time and care is used in extraction, and less trauma inflicted when a local block anesthetic is used than any other anesthetic. A comparison of the work of O'Kell and Elliott<sup>38</sup> and that of Burket and Burn<sup>31</sup> indicates that the incidence of postextraction bacteremias is much lower when local anesthesia is used. Burket suggested that infiltration anesthesia reduced the incidence of bacteremia because of the vasoconstricting adrenaline component of the anesthetic solution. There is no reason to believe that local anesthesia is contraindicated in the group of individuals who are susceptible to subacute bacterial endocarditis, i.e., young and middle-aged persons with valvular damage.

#### CONCLUSIONS

1. Of 92 cases of subacute bacterial endocarditis, extraction was associated with the systemic disease six times.
2. A double relation probably exists between extraction and streptococcal endocarditis. There is evidence from collected reports and from studied cases that postextraction bacteremia causes subacute bacterial endocarditis. Dental extraction may be performed (1) hoping to relieve symptoms of unrecognized subacute bacterial endocarditis, and (2) because bacteremia during streptococcal endocarditis may produce anachoretic pulpitis.
3. One report and one studied case have been presented in which the bacteremia following extraction seems to have caused chronic rheumatic fever to become acute.

4. More detailed histories of patients with streptococcal endocarditis are needed in order to clarify the relation between extraction and endocarditis.

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## EOSINOPHILIC GRANULOMA

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THE clinical and radiographic diagnoses of the following case report was Schüller-Christian's disease, but because of the pathologic report, it is reported as an eosinophilic granuloma.

Eosinophilic granuloma fits in lipoid histiocytosis and is believed by Thoma,<sup>1, 2</sup> to belong in the same general group of diseases as Schüller-Christian's disease. Like Schüller-Christian's disease, it is believed to occur predominately in children. Thoma reports four cases of Schüller-Christian's disease in children<sup>3</sup> and one case in a 51-year-old Italian.<sup>4</sup> He reports one case of an eosinophilic granuloma in a boy 8 years old.<sup>5</sup>

The triad of symptoms, diabetes insipidus, exophthalmus, and defects in the membranous bones found in many of the patients with Schüller-Christian's disease, was present in this patient.

### CASE REPORT

M. T., white, female, aged 28 years.—The patient was referred to one of us (I. S.) and she presented a marked mobility of the lower right first premolar and cuspid teeth. The right lateral and central teeth were not as mobile. There was a cleft with necrotic looking bone in the second premolar to molar region on the ridge. The lingual tissue at the gingiva of the cuspid and lateral teeth was puffed. Sinuses were present but there was no pus visible. There was some discomfort as a result of the mobility of the teeth but no pain. Following the removal of the lower right second premolar the previous year, the patient said that the cuspid and lateral became progressively looser. At the time of removal of the premolar the patient was being treated for diabetes insipidus at the Endocrine Clinic of the New York Hospital. The Wassermann report was negative.

On radiographic examination there was a marked radiolucent area in the mandible extending from the right lateral to the right first molar area (Fig. 1). A radiogram of the skull showed a punched-out radiolucent area in the vault and possible lesions in the occipital area (Fig. 2). During the operation it was noted that the buccal and lingual plates of bone were destroyed at the right premolar area. There was no bone around the cuspid and premolar roots. The area of destruction was filled in with what appeared to be lipoid tissue. The tissue on the lingual side of the lateral and central incisors where the mucous membrane was puffed contained fatlike tissue beneath the mucous membrane. The clinical diagnosis was Schüller-Christian's disease.

Part of the removed tissue was sent to the New York Hospital for examination and diagnosis. The specimen was misplaced. A personal communication

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**Fig. 1.**—Radiograms showing marked radiolucent areas in the right mandible.



Fig. 2.—Radiograms of the skull showing punched-out radiolucent areas in the vault and occipital regions.

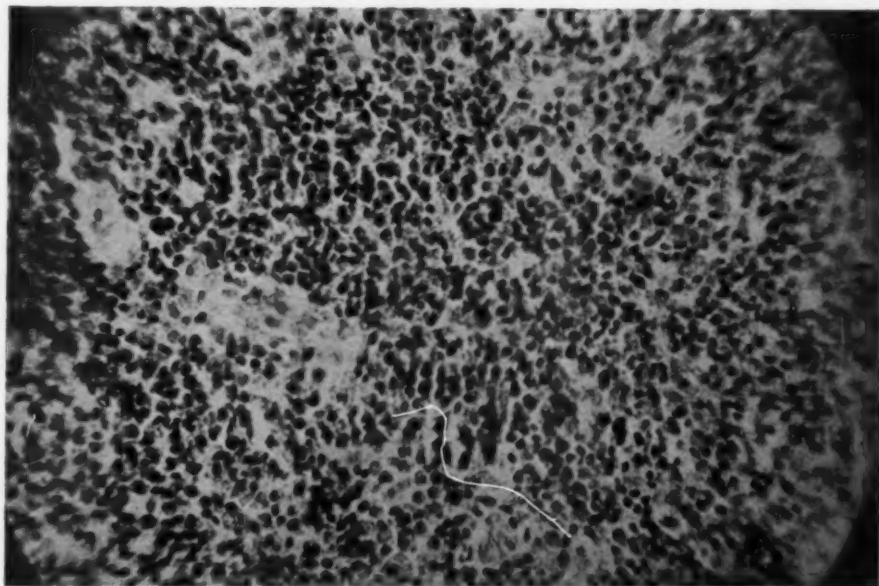


Fig. 3.—Biopsy from mandible.

from the Rockefeller Institute for Medical Research stated that "from the gross characteristics of the tumor there can be little doubt but that this is a Hans Schüller-Christian's disease and that the patient will probably be treated with roentgen therapy." The same diagnosis of Schüller-Christian's disease was made at the Endocrine Clinic of the New York Hospital from radiograms of the skull.

The histological tissue report of the biopsy was: "Tissues are flecked with inflammatory cells which resemble or actually are eosinophiles. The sub-epithelial tissues are unevenly fibrous. The process is suggestive of neoplasm with a strong leaning towards metastatic tumor." (C. G. D.)

We were able to observe the outcome of the intraoral wound in this patient for one year. At the end of that time, the wound had apparently healed. No further data were available as all contact with the patient was lost.

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## MARGINAL PERIODONTITIS

HENRY M. GOLDMAN, D.M.D.\*

THE purpose of this report is to demonstrate the clinical and pathologic findings in one type of disturbance of the periodontium, namely marginal periodontitis, a disease characterized by inflammatory changes of the marginal supporting structures of the teeth.

### DIAGNOSIS

Marginal periodontitis is an inflammation of the marginal periodontium with resorption of the crest of the alveolar bone. The tissues affected are the gingiva, the crest of the alveolar bone, and that portion of the periodontal membrane above and adjacent to the alveolar crest. Although the marginal periodontium of the entire dentition is usually affected, in certain instances the disease may be localized to one or two areas. This is dependent on the causative factors; for example, should the contact point between two teeth be faulty with resultant food impaction, a single interdental area would be affected.

One of the first signs of this disease is a loss of uniform coloration and a thickening of the gingival margin as a result of inflammation. The crevices are for the most part shallow or moderately deepened and may encircle the tooth or may be limited to a single surface. Large quantities of supra and subgingival calculus are observed and a purulent exudate may be found in some cases. The gingivae are soft and spongy and bleed easily; marked recession may be evident.

Fig. 1 shows the typical changes in this disease. Discoloration of the interdental papillae with a purulent exudate exuding from the gingival crevices is evident. The gingival margins are ragged and show evidence of ulceration; this is especially marked in the mandibular right central incisor area. While there is minimal supracalculus present, heavy subgingival deposits are found. In spite of the severe inflammatory process, the teeth are firm; there is marked recession of the gingivae.

In the later stages of the disease, the interdental papillae may tend to become bulbous in appearance and assume a purplish-red color. Calculus and food detritus may fill the interdental spaces. After a considerable loss of alveolar bone has taken place, deep pockets may be found. In this stage the teeth may be slightly or quite loose depending on the amount of remaining root surface embedded in the bone. Once the clinical crown is greater than the clinical root, occlusal conditions may cause traumatism and further loosen the tooth.

Radiographic study of this disease discloses that alveolar resorption proceeds apically from the alveolar margin of the jaws and progresses slowly and gradually in a horizontal direction without the production of vertical bone destruction or enlargement of the periodontal space. A characteristic finding is a cuplike resorption of the interdental crest; this serves to differentiate this disease from senile atrophy and gingivitis. Fig. 2 is a radiograph of the

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mandibular premolar and first molar area in a case of advanced marginal periodontitis. Arrows point to the resorptive process of the alveolar crest; this appears as a cuplike area, the base pointing apically. Thus it is the supporting bone that is first attacked, the alveolar bone, or lamina dura being resorbed later. This radiograph may be compared to the photomicrograph in Fig. 9 which shows the cuplike resorption of the supporting bone. In the late stages of the disease the supporting bone of certain teeth may be affected more than others; this is dependent on occlusal conditions.



Fig. 1.—Marginal periodontitis.



Fig. 2.—Radiograph of mandibular premolar area in a case of advanced marginal periodontitis.

Since the clinical characteristics of marginal periodontitis are essentially the same as gingival inflammations, most investigators believe them to be different stages of the same disease. It is thought that if a gingivitis persists for a long period, it will terminate as a marginal periodontitis. This is probably true, but the fact must be explained why in certain instances a gingivitis will remain as such for very long periods of time, while cases of early gingival

inflammation may be accompanied by loss of the alveolar crest. Correlation of clinical and radiographic findings shows that the severity of the gingival inflammation is not an index of the degree of bone resorption.

#### PATHOLOGY

Microscopic examination of cases of marginal periodontitis discloses that the gingiva and the crest of the alveolar bone are affected.

*Gingival Changes.*—In the gingiva both the epithelium and gingival corium show evidence of inflammatory changes. The epithelium proliferates into the submucosa in fingerlike projections which frequently anastomose and enclose bits of inflamed connective tissue. Inflammatory cells are found in the epithelium. The epithelial lining of gingival crevice is ulcerated allowing either a purulent or serous exudate to be discharged from the inflamed connective tissue of the corium; often the entire crevice may be filled with this discharge. The type of exudate is dependent upon whether the serous or the cellular elements predominate. Thus the amount of purulent discharge from a pocket depends on the degree of destruction of the crevicular epithelium and the amount of cellular infiltration of the gingival corium. The epithelial attachment is usually intact.

Fig. 3 is a photomicrograph of the interdental gingiva in a case of advanced marginal periodontitis. The crevices are moderately deepened and are filled with calculus and a purulent discharge. The epithelial retia project deeply into the submucosa in several places, anastomosing with one another. At *A* the epithelium is missing; the floor of this ulcerated area is composed of the densely inflamed connective tissue of the gingival corium. The space marked *Ar* is artifact created during processing of the tissue.

In the changes in the gingival corium the outstanding feature is an inflammatory cell infiltrate which is so dense at times that very little stroma is evident. Numerous lymphocytes, plasma cells, and histiocytes are found. This infiltrate extends deeply into the marginal periodontium following along the course of the blood vessels, and separating the connective tissue bundles of the transeptal fibers. These fibers which pass just above the crest of the bone are seen in all stages of the disease. Usually several sets are present, new ones being formed as those incisally are destroyed. Inflammatory cells are found adjacent to the alveolar crest and in some instances in the marrow spaces of the interdental bone.

Fig. 4 is a photomicrograph of the interdental tissues between the mandibular right premolars, showing the pathologic processes seen in marginal periodontitis. The crevices (*C*) are very shallow although there is marked inflammation of the gingival corium (*I*). Several sets of transeptal fibers are present; inflammatory cells are seen between the bundles (*TS*). Resorption of the bone of the alveolar crest is evident at *R*. Thus, although the crevices are shallow, resorption of the crestal bone is seen. This demonstrates that the depth of the crevice does not influence the condition of the alveolar crest.

The inflammatory infiltrate is usually present in close proximity to the epithelial attachment and in between the adjacent bundles of connective tissue



Fig. 3.—Interdentary gingiva in marginal periodontitis. *T*, tooth; *Ca*, calculus; *Ex*, inflammatory exudate; *E*, crevicular epithelium; *A*, ulceration of crevicular epithelium; *EA*, epithelial attachment; *I*, inflammatory cells; *Ar*, artifact.



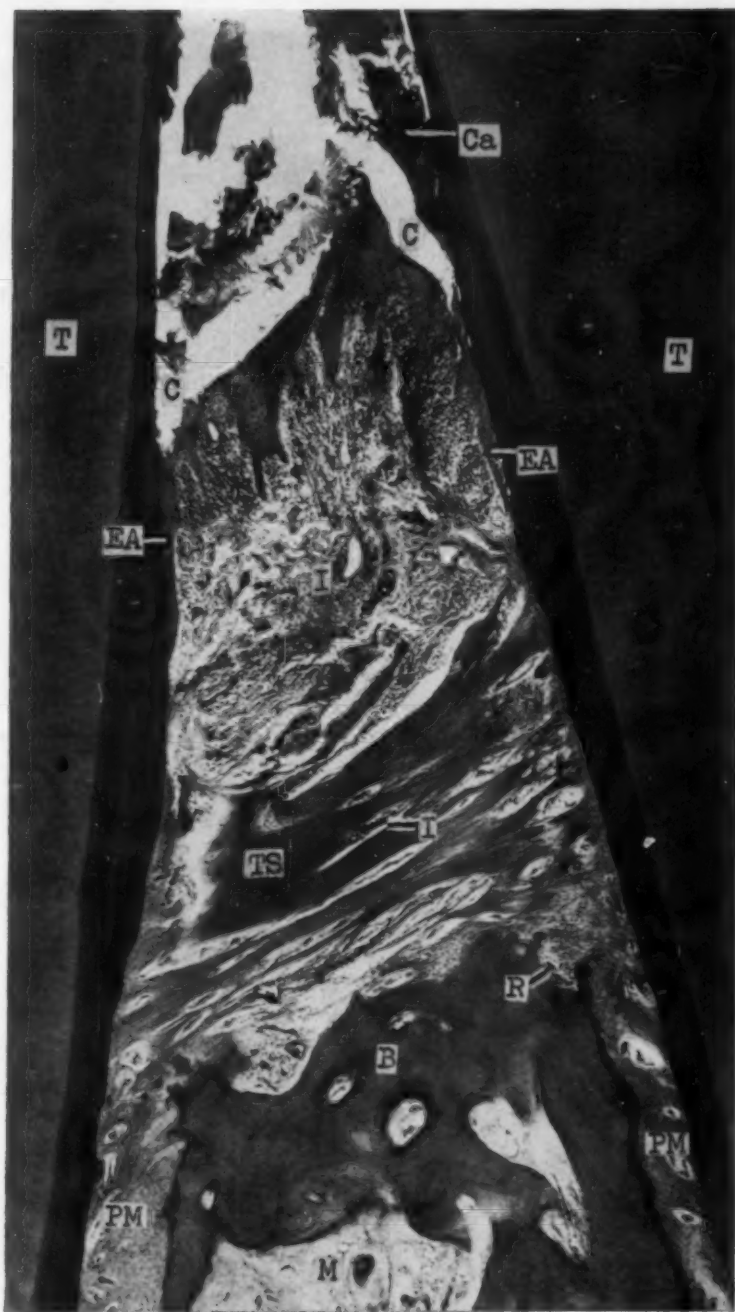


Fig. 4.—Interdental tissues in marginal periodontitis. *T*, tooth; *Ca*, calculus; *C*, gingival crevice; *EA*, epithelial attachment; *I*, inflammatory cells; *TS*, transseptal fibers; *B*, bone; *R*, resorption; *PM*, periodontal membrane; *M*, marrow.

of the periodontal membrane. Fig. 5 shows such a condition. The epithelial attachment is seen at *EA* and the adjacent periodontal fibers at *PM*. The inflammatory cells (*I*) are chiefly lymphocytes and plasma cells.

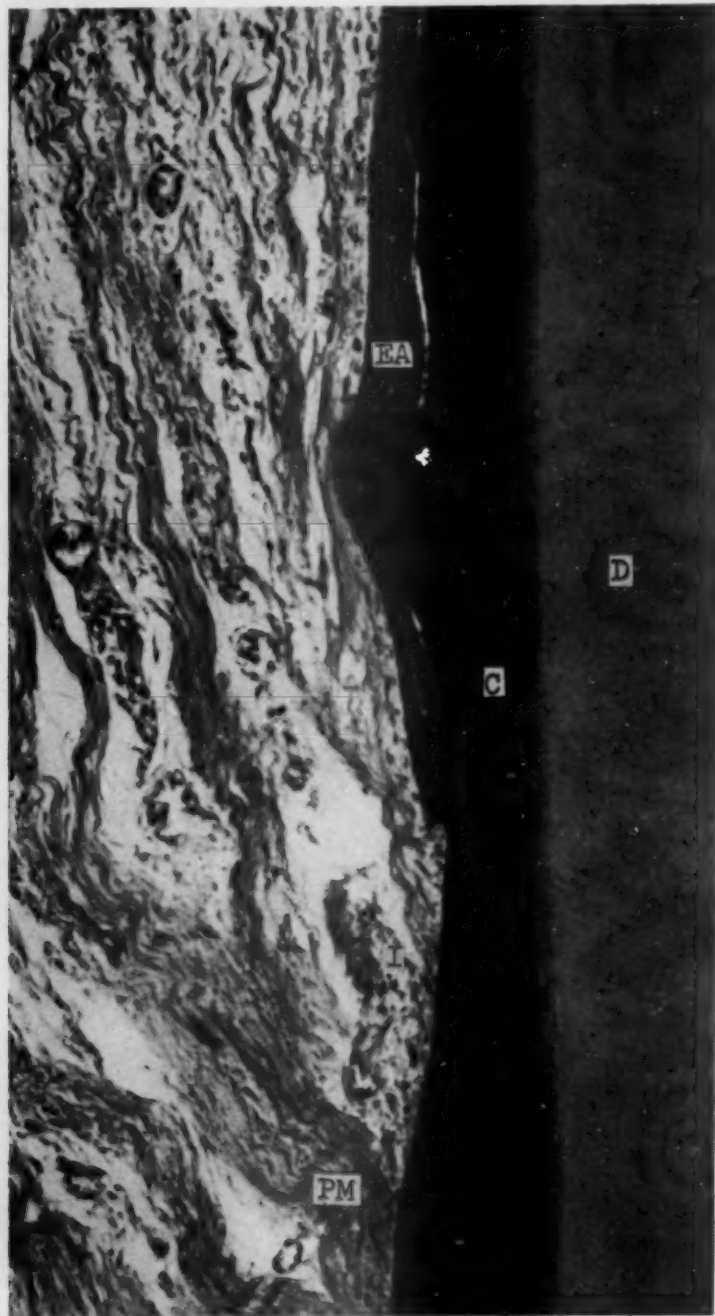


Fig. 5.—Epithelial attachment and adjacent periodontal fibers in marginal periodontitis. *D*, dentine; *C*, cementum; *EA*, epithelial attachment; *PM*, periodontal fibers; *I*, inflammatory cells.

The periodontal membrane, however, is almost always spared except that portion just described. Fig. 6 is a photomicrograph showing a severe inflammation in the gingival corium and to a much lesser degree in the marrow space

of the interdental bone below the crest. There is no inflammatory infiltrate in the periodontal membrane proper. Rarely, however, one does find inflammatory cells extending directly from the gingiva into the periodontal membrane proper. Fig. 7 is a high-power photomicrograph with a low-power insert showing such a condition.



Fig. 6.—Interdental tissues in marginal periodontitis. T, tooth; E, enamel space; C, gingival crevice; A, bottom of gingival crevice; EA, epithelial attachment; I, inflammatory cells; TS, transseptal fibers; PM, periodontal membrane; B, bone; M, marrow.

*Relationship of Calculus to the Gingival Crevice.*—Lying on the gingiva or extending into the gingival crevice one finds calculus in almost all sections studied. At times the gingival wall is distended and is not in contact with the calculus, while in other instances the calculus fills the entire crevice. The former

probably is due in many instances to shrinkage during processing of the tissue. Regardless of the position of the calculus, the crevicular epithelial lining is usually ulcerated and sometimes completely destroyed, the denuded surfaces consisting of inflamed connective tissue. Fig. 8 shows calculus entirely occupying deep narrow pockets and lying in apposition with the crevicular epithelium; ulceration of the epithelium is apparent.



Fig. 7.—High-power photomicrograph of inflammatory cells in the periodontal membrane, with a low-power insert for orientation. *D*, dentine; *C*, cementum; *I*, inflammatory cells; *PM*, periodontal membrane; *B*, bone.



*Bone Changes.*—The bone changes are important in that they are the differential features in distinguishing this disease from gingivitis. The alveolar crest can be seen undergoing lacunar resorption; Howship's lacunae, in which osteoclasts may be seen, are present. Examination of the bone cells in this area demonstrates that they are viable.

Resorption of the alveolar crest takes place medially, first the supporting bone being destroyed and then the lamina dura. This results in a cuplike notch



Fig. 8.—Calculus in deepened gingival crevices with ulceration of the crevicular epithelium in marginal periodontitis. *C*, cementum; *Ca*, calculus; *Ce*, crevicular epithelium; *U*, ulceration; *I*, inflammatory cells.

in the alveolar crest (Fig. 9), a finding which can be correlated in the radiographic examination of this disease. The marrow spaces are usually fibrosed, probably because of the presence of inflammation. In the later stages of the disease when the height of the alveolar bone is reduced, traumatism may occur.

*Relationship of the Gingival Crevice to the Alveolar Crest.*—The study of sections of jaws discloses that neither the depth of the gingival crevice nor the degree of inflammatory cell infiltrate are directly related to the condition of the

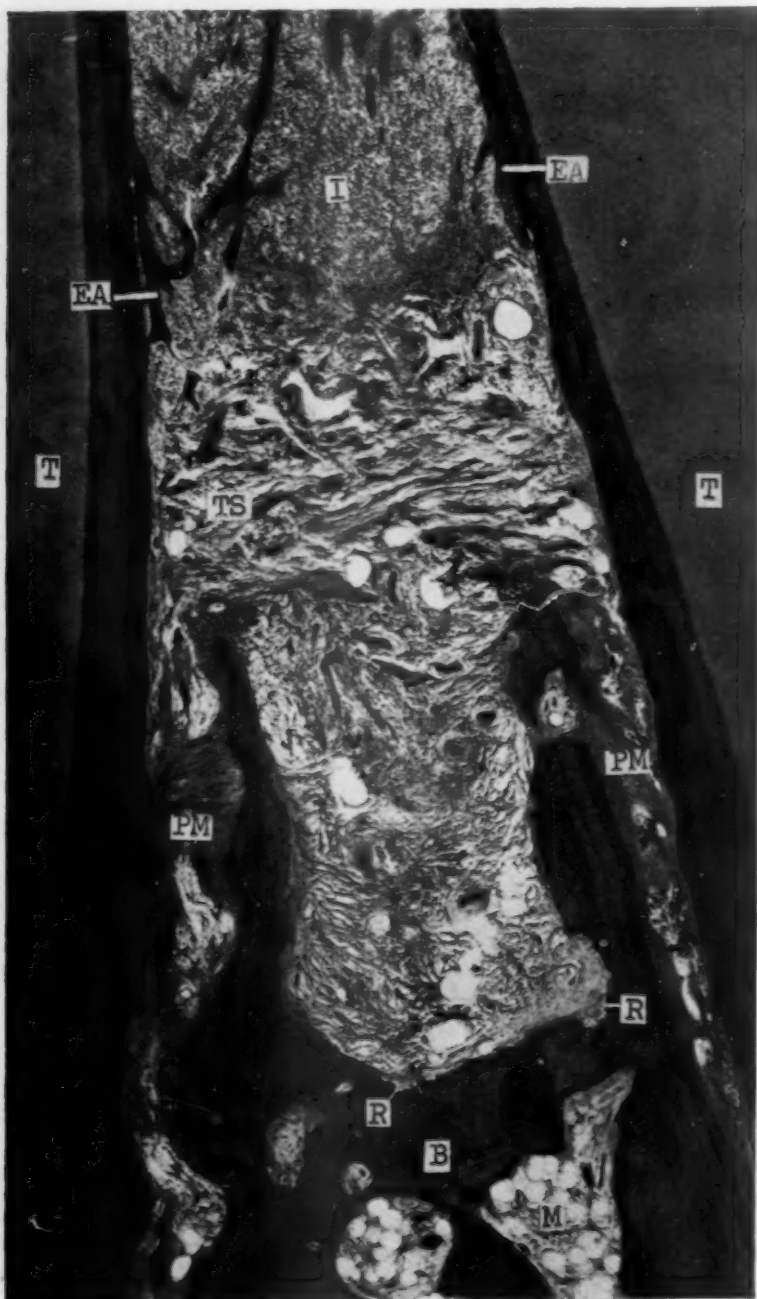


Fig. 9.—Resorption of the interdental bone in marginal periodontitis. T, tooth; EA, epithelial attachment; I, inflammatory cells; TS, transseptal fibers; PM, periodontal membrane; B, bone; R, resorption.

alveolar crest (Figs. 4 and 6). Sections showing shallow or deepened gingival crevices may show either resorption of the alveolar crest or an apposition of bone; often a state of inactivity is found. This substantiates the clinical findings that some cases of gingivitis do not show evidence of alveolar crest resorption radiographically after many years of the disease.

## SUMMARY

Marginal periodontitis is a disease of the marginal periodontium characterized by inflammation and resorption of the alveolar crest. Clinically it must be differentiated from gingivitis and senile atrophy. Although this disease is associated with calculus in the gingival crevice and inflammation of the gingiva, study of human autopsy material reveals that neither the depth of the gingival crevice nor the degree of the inflammation bear any relationship to the condition of the alveolar crest. Why marked resorption is seen in some cases and not in others cannot be established from histologic evidence.

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THE JAWS IN PAGET'S DISEASE (OSTEITIS DEFORMANS): WITH  
SPECIAL REFERENCE TO OSTEOPOROSIS  
CIRCUMSCRIPTA CRANII

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INTRODUCTION

OSTEITIS deformans of the maxilla and mandible may occur as the initial lesion or appear as one of the stages during the course of Paget's disease. There is evidence that a primary abnormal vascularity, followed by an advancing sclerosis and ending in diffuse sclerosis<sup>1</sup> is part of the complicated picture of Paget's disease.

These sclerotic changes may occasionally be seen in the maxilla and mandible but are not always recognized as part of Paget's disease. Very often, these lesions are interpreted as early osteitis fibrosa localisata, or as classified by Thoma,<sup>2</sup> one of the group of the fibro- or fibrous osteomas. These early lesions are difficult to differentiate clinically, roentgenologically and, at times, histopathologically.

DEFINITION

Briefly, Paget's disease is a disease of bone occurring in middle age, characterized by lesions of bone destruction and proliferation in sequence<sup>3</sup> resulting in eventual enlargement of the head and jaws, bowing of the tibiae, and involvement of scattered areas in many of the other bones.

THEORIES

The etiological factor is unknown. A number of theories have been expounded, none of which answers the questions which may arise in the course of the disease.

The endocrine system, bacteria, intestinal intoxication, neurotrophic changes, and intoxication by mineral acids have been discussed and found wanting. Knaggs, who made an extensive study of Paget's disease was inclined toward a theory advanced by Sir Jonathan Hutchinson,<sup>4</sup> that "toxins due to faulty tissue metabolism or of intestinal origin may be at least of some importance etiologically."

SYMPTOMATOLOGY

The objective signs are the gradual enlargement of the skull, frequently of the maxilla, more rarely of the mandible, bowing of the tibiae, retraction of the chest wall, and shortening of the stature.

Subjectively, the patient may complain of vague pain in the bones, joints, and muscles. As the bones of the skull and face become involved, paranasal sinus discomfort and occasional atypical neuralgic pain in the jaws manifest themselves.



Because of the complaint of occasional vague body pain, the patient is often sent to the dentist for the elimination of possible dental foci of infection. Routine x-ray examination may reveal changes in the mandible and maxilla significant of possible beginning Paget's disease, necessitating further studies.

#### HISTOPATHOLOGY

Normally, bone resorption and bone proliferation take place, the amount depending on the stresses exerted on the bones. Rifenstein and Albright<sup>3</sup> have shown that the initial bone lesion in Paget's disease is a destructive one and that bone destruction and bone formation are very marked. As the new bone is laid down, cement lines mark the junction, giving the bone structure the appearance of a mosaic pattern microscopically, which is pathognomonic of this disease. (Fig. 1.)

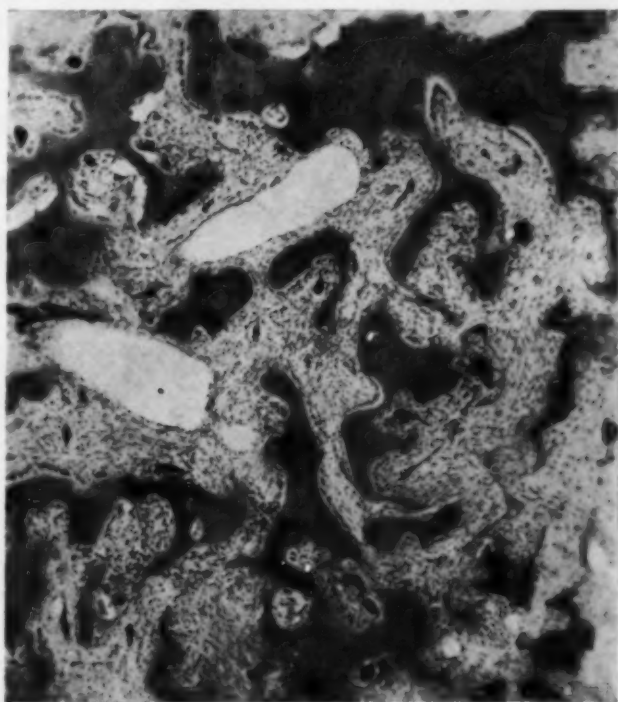


Fig. 1.—Mosaic pattern in Paget's disease. Bone taken from case shown in Fig. 4.

The new bone is fibrous and of very fine porous character. New formations may be seen as fibrous or osteoid tissue in varying stages.

With the great amount of destruction which takes place, the number of osteoclasts which may be seen are fairly small.

#### CLINICAL SIGNIFICANCE OF X-RAY FINDINGS

Paget's disease may be conceived as occurring in stages. Early in the disease the bone destruction is greater than the bone proliferation. This manifests itself as an osteoporosis undergoing dissolution, giving an appearance similar to what may be seen in the osteitis fibrosa generalisata of hyperparathyroidism.



Fig. 2.—"Cotton wool" appearance in Paget's disease. Note size of jaws.



Fig. 3.—The skull in advanced Paget's disease.

The dissolution may be sufficiently extensive in localized areas of the calvarium and jaws to resemble the cysts of hyperparathyroidism in the x-ray film. Many times the differential diagnosis entails careful examination of the values of the blood serum calcium, phosphorus, and phosphatase.

In hyperparathyroidism, the blood serum calcium is increased and the phosphorus decreased. In Paget's disease, the calcium and phosphorus are within normal limits or low. The phosphatase value is increased in both conditions, but is generally greater in Paget's disease.

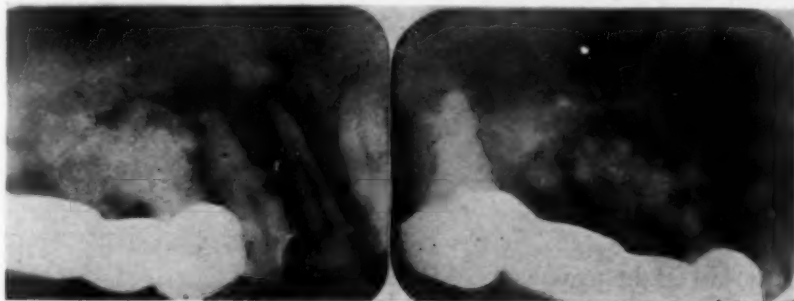


Fig. 4.—The maxilla in Paget's disease. Bone destruction and proliferation going on simultaneously.



Fig. 5.—The mandible in Paget's disease. Note width of mandible and sclerotic appearance of the bone.

As the condition progresses and bone proliferation becomes more marked, resulting in the enlargement of the skull, the x-rays reveal the characteristic "cotton wool" appearance. (Figs. 2 and 3.) The maxilla reveals both bone destruction and proliferation going on at the same time. Areas of diffuse radiopacities alternate with radiolucencies. (Fig. 4.) When the mandible is involved, the bone is greatly hypertrophied, both radiopaque and radiolucent areas being evident in the medullary portion of the bone also. (Fig. 5.)

## OSTEOPOROSIS CIRCUMSCRIPTA CRANII

It has been shown that, occasionally, a patient presents himself whose x-rays reveal circumscribed radiolucencies in the calvarium of the skull and a marked osteoporosis of the jaws. These cases, when followed for years, eventually develop typical Paget's disease.<sup>5</sup> Thoma<sup>6</sup> quotes Schüller, Weiss, and Kasabach and Dyke as having pointed out that this picture is an early stage, or the so-called monophasic stage of Paget's. They and other authors have described these lesions under the title of osteoporosis circumscripta cranii, a case of which is being reported.

## CASE REPORT OF OSTEOPOROSIS CIRCUMSCRIPTA CRANII

J. H., a white man, aged 45 years, had always been well. He had recently noted lameness of the shoulders, back, legs, and joints. He began having headaches and pain in the left maxilla. A physician was consulted who referred him for oral examination to rule out possible foci of infection.

Patient stated that the last two times he purchased hats they were a larger size than those bought previously.

Routine dental x-ray examination disclosed many amalgam fillings and inlays. Throughout both jaws were rarefied areas, some diffuse and others circumscribed, especially over the left central, premolar, and molar areas in the maxilla, and in both premolar areas of the mandible. (Fig. 6.) All teeth, except the left maxillary lateral incisor, were vital to electrovitality tests.

Because of this marked osteoporosis, x-rays of the skull, pelvis, and long bones were taken. The right and left os ischii showed marked bony changes, consisting of thickening and sclerosing of the bone and some rarefying changes not quite characteristic of Paget's disease. The skull revealed circumscribed radiolucent areas in the occiput and in the top of the calvarium. (Fig. 7.)

The urine showed no albumin or sugar. Serology was negative. The blood findings were as follows:

Hemoglobin	108 per cent (Sahli)
Red blood cells	6,200,00
White blood cells	6,000
Polymorphs	64 per cent
Lymphocytes	22 per cent
Monocytes	12 per cent
Eosinophiles	2 per cent
Blood calcium	7.6 mg. per 100 c.c. serum
Blood phosphorus	2.6 mg. per 100 c.c. serum

Repeated calcium determinations showed low values. This ruled out osteitis fibrosa cystica (Recklinghausen's disease), especially since the patient was ambulatory and working.

This is a relatively early case of Paget's disease. If it is true that osteoporosis circumscripta cranii is a monophasic stage of Paget's disease, the bone changes that take place in the next few years will be noted.

The next stage should show an overproduction of osteoid tissue in the skull undergoing some calcification and giving the "cotton wool" appearance of the case shown in Fig. 4. As the condition progresses, the thickness increases and the bone pathology becomes more marked, as in Fig. 5.





Fig. 6.—Osteoporosis of maxilla in osteoporosis circumscripta cranii.



Fig. 7.—The skull in osteoporosis circumscripta cranii. Note marked radiolucency in occiput and anterior calvarium. Note size of jaws.

## SUMMARY

Paget's disease is a progressive disorder of one or more bones, the monophasic stage of which may be osteoporosis circumscripta cranii.

A case is reported which will be placed on observation and x-rays of all the bones taken at intervals to determine and confirm development into typical osteitis deformans.

In the jaws, osteosclerotic appearing lesions may be the first evidence of Paget's disease, necessitating x-ray studies of the skull, long bones, and pelvis.

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311 COMMONWEALTH AVENUE

## APPLIANCES FOR EXTERNAL FIXATION OF THE MANDIBLE AND CRANIAL FIXATION OF THE MAXILLA

JOHN MARQUIS CONVERSE, M.D.,\* NEW YORK, N. Y.

THE appliance for external fixation of the mandible illustrated (Fig. 1) was made for the use of the French Army in North Africa and Italy. The appliance was designed in collaboration with Brig. Gen. A. Romey of the French Army Medical Corps. It is a modification of appliances previously used, but is smaller and lighter. The appliance presents a jackscrew (Fig. 1) which permits variations of the distance between the twin pin-units. Lateral variations of each pin-unit are also possible (Fig. 1) as in the first appliance utilized.<sup>1</sup> The small universal joints are modifications, in a smaller size, of the joints utilized by Clouston and Walker.<sup>2</sup>

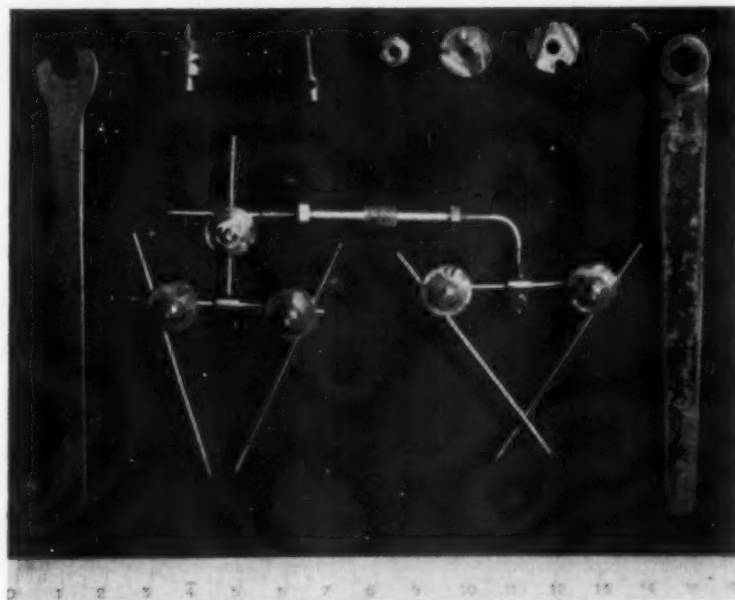


Fig. 1.

Universal joints of the external fixation appliance can be employed for cranial fixation of the fractured maxilla (Fig. 2). Kirschner wires are used as connecting rods between the plaster head cap and the maxillary splint. Kirschner wires are found in the orthopedic department of any hospital and come in variable sizes. They are made of stainless steel and present a certain elasticity.

\*Major, French Army Medical Corps, Consultant in Plastic Surgery; at present in the Army of the United States.

This elasticity can be used to reduce impacted fractures. The Kirschner wires are put in place and curved, and the elasticity of the wire produces the desired displacement of the maxilla downward and forward.

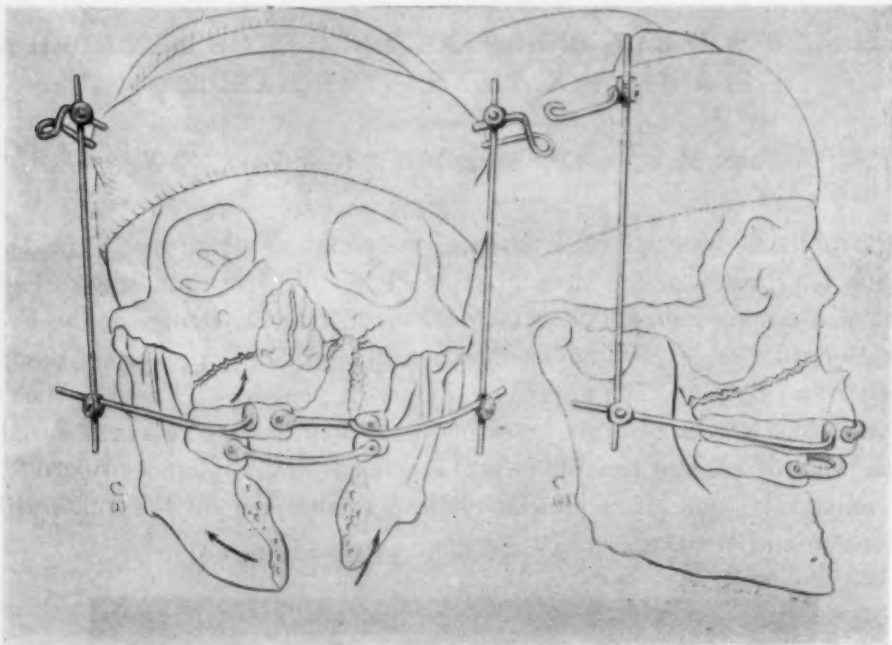


Fig. 2.

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## Case Reports

### CASE NO. 92

#### EXTENSIVE CAVITATION IN THE MANDIBLE, SIMULATING A CYST

MATTHEW LOZIER, B.S., D.D.S.,\* NEW YORK, N. Y.

THIS case is being reported primarily because of its uncommon character, which was revealed during the time of the operation. While the patient's history, and clinical, laboratory, and radiographic examinations led to a tentative preoperative diagnosis of either an odontogenic or traumatic cyst, the condition I was dealing with proved instead an enormous cavitation possibly due to some progressive osteolytic process originating, in all probability, from a pulpless tooth in the involved region.

The patient, a young man 16 years old, looked well developed for his age and was apparently in fine physical condition. Upon completion of high school he had obtained employment as a shipping clerk, doing quite heavy physical labor. He presented himself for a clinical and radiographic examination of his teeth with the intention of having us remove one of his upper teeth and the lower left first molar, which were beyond saving. The patient was not in pain and had recently returned from his dentist, who performed all of the most essential operative work.

Our complete dental intraoral radiographic examination disclosed a radiolucent area situated below the left lower second premolar, first and second molars, and extending distally over the unerupted and still incompletely calcified third molar. An extraoral radiograph of the left mandible (Fig. 1) disclosed a large well-defined radiolucent area extending close to the necks of the afore-mentioned teeth and involving practically the entire width of the body of the left jaw. The radiolucency appeared to be of a fairly uniform character with no evidence of trabeculation. Palpation of the cortical plates of the jaw revealed neither crepitation nor bending. The examination for vitality elicited normal response in the lower left first premolar, no response in the adjoining second premolar, and very slight response in the second molar. The upper jaw and the right mandible were found clinically and radiographically entirely normal. There were no signs of facial deformity or any subjective symptoms. There was no history of uncommon childhood illnesses or of inflicted trauma, though careful questioning revealed admission of sustained blows while boxing. The lower left first molar, according to the patient, was never annoying, though it had been slowly decaying for the past several years.

The patient's parents were active and apparently in good health, but his older sister was in somewhat "delicate health," sustaining two fractures of her

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Fig. 1.



Fig. 2.

long bones, one while "she lifted her leg walking up a steep hill." This sister was treated for her injuries in a hospital and the history of her case was unobtainable.

To rule out the possibility of dealing here with a tumor of nonosteogenic or nonodontogenic in origin or some skeleton disease affecting the jaws, the patient was subjected to a complete hematological examination, including blood chemistry, as well as a careful urinalysis and radiographic examination of the long bones. No positive findings were obtained from all these examinations and a tentative diagnosis of either an odontogenic or a traumatic cyst was made.

The operation was performed under block anesthesia. Two short vertical incisions, one leading from the alveolar crest to the mucobuccal fold over the distal aspect of the third molar, and the other over the mesial aspect of the first premolar were made. These were joined with a long horizontal incision over the alveolar ridge parallel with the necks of the teeth. The mucoperiosteum was then laid back and the exposed buccal plate, when examined, was found entirely sound in appearance. The third molar was exposed, and this tooth and the adjoining second and first molars and the second premolar were carefully extracted. The first molar was found to have three fully developed roots. Upon examination of the interior aspect of the jaw after the teeth were removed, a large cavitation, with the mandibular vessels lying in close proximity to its bottom and suspended in space, was observed. There was absolutely no evidence of cystic contents, membrane, or even a trace of a lining, nor was there any fluid or cholesterol crystals. The medial aspects of the buccal and lingual plates were entirely normal in appearance, though the buccal plate seemed to be very thin. During the entire surgical procedure the bleeding was moderate and no pain was experienced by the patient, except when the second and first molars were luxated and the mandibular nerve was touched. Before suturing the lateral flaps, the bottom of the cavity was dusted with sterile sulfanilamide powder over which some sulfadiazine guaiacol-glycerin paste was deposited. A strip of iodoform gauze moistened in guaiacol-glycerin was placed loosely over the paste.

When the patient was examined on the following day, he commented that there was slight tenderness in the left first premolar upon pressure. The tooth was again tested for vitality and found normal. No paresthesia or bleeding was experienced by the patient at any time after the operation. Healing took place rapidly and uneventfully and dressings were discontinued after a two-week period. The submitted postoperative radiograph (Fig. 2) was taken about three weeks after the operation.

As to the nature of the cavitation, there is a remote possibility that at some time prior to the operation a cyst was present in the jaw, which, for some reason or other, changed its character and consistency and was consequently completely destroyed, leaving in its place the present cavitation. It does not seem likely, however, that Nature would tolerate for any length of time a large abnormal cavity in the bone without attempting to do away with it in one manner or another.

## Editorial

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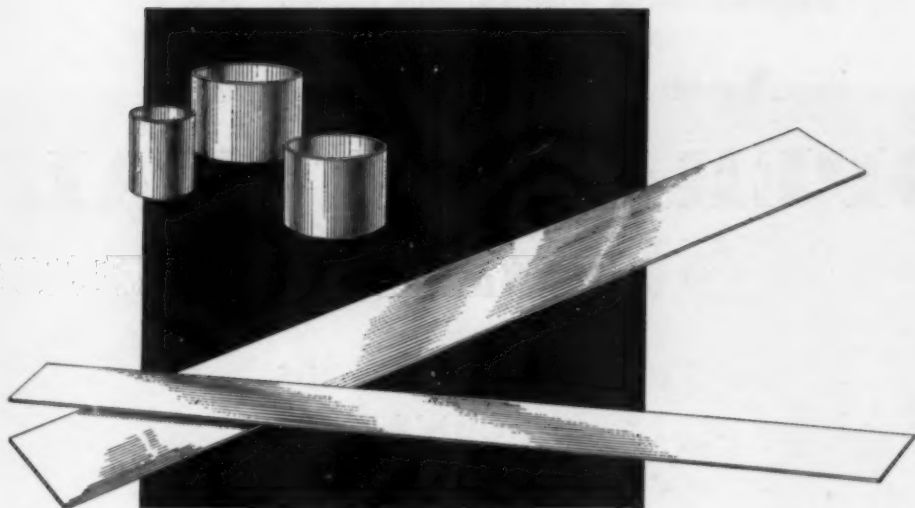
### The Patient's General Health

We are presenting this month a review of the relationship of endocarditis to dental procedures, written by Dr. W. F. Barnfield. Reviews of other medical subjects important because of their dental relationships will be published in the future. We feel that it is necessary that the oral surgeon be familiar with topics of modern internal medicine, such as heart disease, blood dyscrasias, diabetes mellitus, and many other diseases which may greatly affect the risk of anesthesia and the outcome of surgical procedures. This effort does not intend to make readers experts in diagnosis or treatment of such diseases, but we feel strongly that all dental and oral surgeons should have sufficient knowledge of general medical problems that they may safeguard the welfare of patients while in their charge.

Because of the autonomous method of education in dentistry, many of us forget that the teeth and supporting structures are part of the body, and we fail to inquire sufficiently about the family and personal history of patients or the state of their general health to discover important signs and symptoms that would alter the prognosis and change the procedures of treatment. In general surgery, supportive treatment is considered of great importance, both in preparation for an operation and in speeding the recovery. As oral surgeons, we owe it to our patients to be able to recognize symptoms of internal diseases, so that we may procure the advice and help of the patient's physician, to obtain the best possible result.

*K. H. T.*





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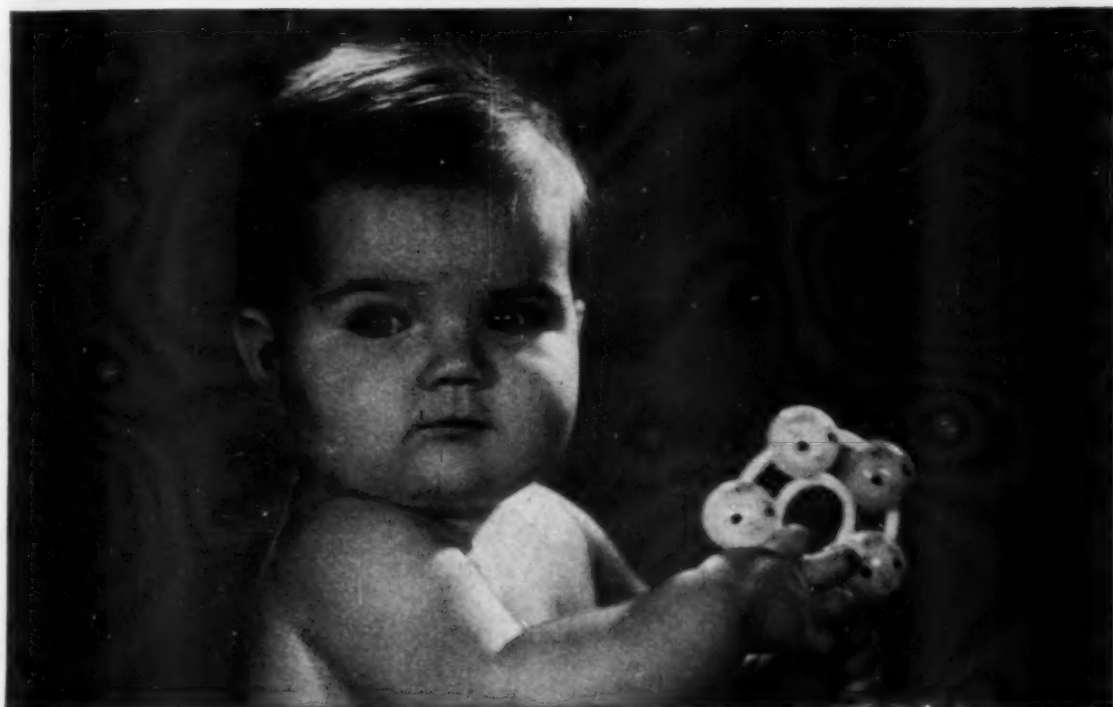
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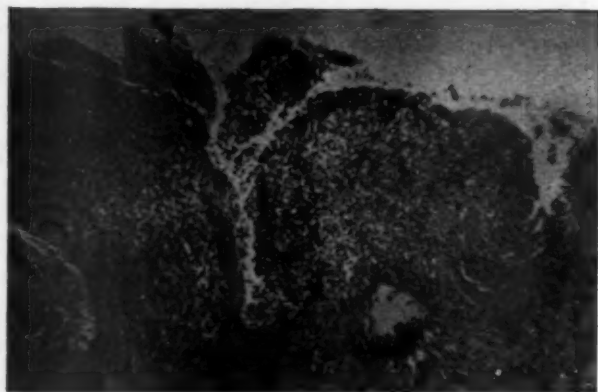
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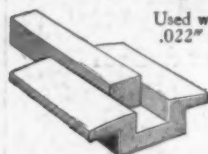
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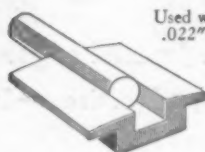
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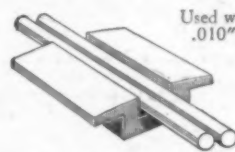
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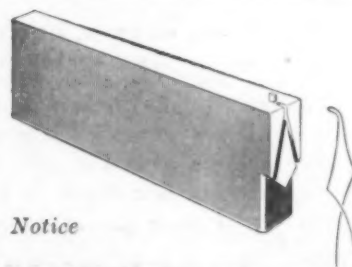
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# Army Medical Museum Number

## FATAL INFECTIONS OF THE CENTRAL NERVOUS SYSTEM AND MENINGES AFTER TOOTH EXTRACTION

WITH AN ANALYSIS OF TWENTY-EIGHT CASES

CAPTAIN WEBB HAYMAKER, M.C.\*

### INTRODUCTION

OVER the past years the Army Institute of Pathology has received twenty-eight cases in which fatal intracranial or spinal lesions occurred after tooth extraction. Fourteen of them have reached the Institute since Pearl Harbor. In view of the 125,000 accessions at the Institute, of which approximately 65,000 are autopsies, this type of complication is uncommon. There were four other instances of spread to the intracranial cavity after tooth extraction but they were not included in this series because acute suppurative osteomyelitis of the jaw was advancing rapidly at the time of extraction. This communication is concerned only with cases in which infection ultimately gaining entrance to the intracranial cavity or the spinal cord, was initiated or precipitated by tooth extraction.

The reasons for extraction were several: periapical abscess disclosed by roentgenogram, 7 (of which 2 were associated with caries); caries, 3; impaction, 2; toothache, "painful jaw," malposition of teeth, painful eruption, periodontoclasia, "edema of gums," and chronic osteomyelitis, 1 each; and not specified, 9. Local infiltration anesthesia with procaine was employed in 7 cases, conduction (nerve block) in 4; in the 17 remaining the mode of anesthesia is not known.

A review of the data on the condition of the mouth at the time of tooth extraction revealed that the stage was set for the ultimate outcome in eight of the cases, as there was Vincent's infection, periodontoclasia, and other forms of oral sepsis; in the remainder the mouth was clean. Chronic disorders come into consideration in only five of the cases: they include arthralgia, coronary disease, prostatitis, alcoholism, paranasal sinusitis, and nasomaxillary polyposis. The ages of the patients ranged between 20 and 52, the average being 33.

The intracranial complications of tooth extraction varied widely not only as to type but also in the manner of spread of the infection and in duration of the illness. Disregarding a certain degree of overlap in the pathologic findings, the cases fall into the following categories:

Subdural empyema	1
Subdural empyema and brain abscess	2
Leptomeningitis	2
Leptomeningitis and brain abscess	2
Suppurative encephalitis and ependymitis	1
Brain abscess	8
Sinus thrombosis	11
Transverse myelitis	1

\*Army Institute of Pathology, Army Medical Museum, Washington, D. C.

## CASE HISTORIES

There are numerous cases on record in which cellulitis extended from the region of tooth extraction along fascial planes to the base of the skull where the infective process was halted by prompt surgical intervention; but in most of the cases that follow, the infective process proceeded upward relentlessly despite combative measures, taking one or more routes to traverse the skull. In the ensuing pages an effort is made to trace the paths of spread from offending tooth sockets to the intracranial cavity.

## SUBDURAL EMPYEMA

In the first case the infective process penetrated the base of the skull and the dura, extending widely in the subdural space and leading to multiple abscess formation. The arachnoid proved an effective barrier to further spread.

**CASE 1** (AMM Accession 92742).—Male, white, age 26. *Cellulitis of the upper jaw, left, with development of subtemporal and pterygoid abscesses; sphenoidal sinusitis; osteomyelitis of the greater wing of the sphenoid, and the squamous portion of the temporal bone, left; subdural empyema, bilateral; early bronchopneumonia.* Death occurred 36 days after tooth extraction.

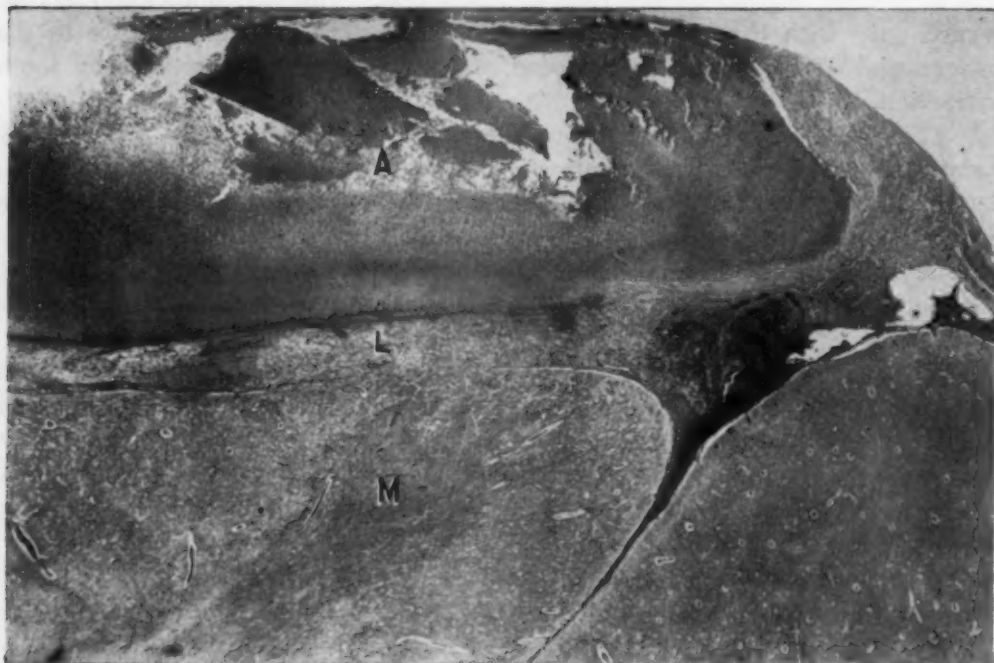


Fig. 1.—Subdural abscess of inferior surface of brain. The abscess (A) within the subdural space is well circumscribed. The underlying leptomeninges (L) are thickened and contain relatively few inflammatory cells. In one region a wedge of cerebral cortex has undergone malacia (M). AMM Neg. 83441. Masson's trichrome stain.  $\times 14$ .

**Clinical Data.**—On 20 October, 1942, the following teeth were removed from the left maxilla: second bicuspid, and the first, second, and third molars (L-5, 6, 7, 8). The lower left second molar (L-15) also was removed. Extraction, performed under local procaine anesthesia, was done because of advanced caries. Several days later, headaches developed and the left side of the face became swollen. The patient was admitted to hospital on 1 November. The temperature was 101.6° F., the pulse 100. The left side of the face, including the left lower



eyelid and the adjoining parietotemporal region, was swollen and tender but not fluctuant. There was a slight discharge from the empty tooth sockets of the left maxilla. The cervical lymph nodes could not be palpated. Sulfathiazole therapy was started, the subsequent blood levels of the drug averaging 5.0 mg. per cent.

On 2 November the gums from L-4 to L-8 were incised but no pus was found. On 5 November an attack of grand mal occurred. Two days later the neck became rigid, and motor aphasia and a paresis of the right side of the face and of the right upper extremity were noted. The pupils were equal in size and responded readily to light and in accommodation. A spinal puncture revealed mild meningitis. During the next two days there were repeated Jacksonian seizures involving the right side of the face and the right arm.

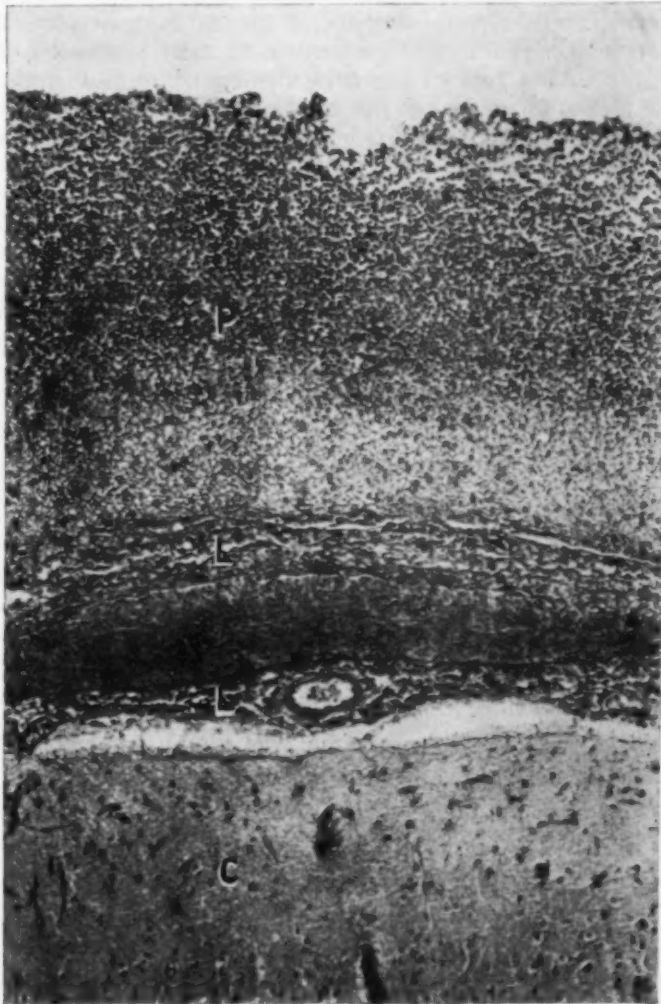


Fig. 2.—Subdural empyema of the lower convex surface of the brain. The pus (P) lies free in the subdural space. The adjacent leptomeninges (L) contain a few inflammatory cells. The cerebral cortex (C) is relatively normal. AMM Neg. 83438. Hematoxylin and eosin stain.  $\times 145$ .

On 9 November an exploratory trephine in the left temporal region revealed subdural empyema: about one ounce of pus was evacuated. On the next day the upper left jaw was incised and abundant pus escaped into the mouth. On 23 November the site of previous trepanation was enlarged, and in

the region of the left frontal pole more pus was found. In an effort to find a cerebral abscess an exploring cannula was inserted into the brain in four planes but none was encountered. Death occurred on 25 November, 36 days after tooth extraction.

*Laboratory Data.*—Blood examination on 1 November, WBC 20,100 (P 89, L 8, M 3); on 4 November, WBC 18,650 (P 76, L 22, M 2). Spinal fluid examination on 7 November, WBC 268 (P 81, L 19); on 22 November, WBC 121 (P 65, L 35); on 24 November, WBC 274 (P 32, L 68). Culture of blood on 19 November, negative; of spinal fluid on 7 and 24 November, also negative. Smears of subtemporal pus disclosed staphylococci; cultures were sterile.

*Pathology.*—Removal of the brain revealed extensive subdural empyema covering all lobes of the brain except the left occipital; the pus was especially abundant over the base of the brain and on the superior surface of the left tentorium cerebelli. In several regions the pus had become well loculated (Fig. 1). Microscopic examination revealed in most sections a minimal leptomeningitis (Fig. 2). Malacia of the superficial part of the cerebral cortex was present in some places (Fig. 1) but not in others (Fig. 2).

When the dura was stripped from the base of the skull it was seen that the floor of the left middle cranial fossa was eroded. The cranial defect was most marked in the region lateral and anterior to the foramen ovale, involving the greater wing of the sphenoid and the squamous portion of the temporal bone. A probe could easily be passed through the eroded bone directly into the oral cavity in the region of the left superior alveolar border and through the eroded sella turcica into the pus-filled sphenoidal sinus. The left subtemporal and pterygoid fossae contained abscesses. There was scanty leptomeningitis but no brain abscess.

Except for early bronchopneumonia, no changes were observed in the thoracic and abdominal organs.

*Comment.*—Judging from the attack of grand mal and the subsequent motor aphasia and paresis of the right side, the infective process reached the subdural space about fifteen days after tooth extraction. Although the induration of the parietotemporal region receded early in the course of the illness there were large abscesses in the subtemporal and pterygoid fossae, which were not recognized until autopsy. In view of the location of the pus extracranially it is not surprising that the greater wing of the sphenoid and the sella turcica bore the brunt of the cranial invasion.

The diagnosis of subdural empyema was made during life, and drainage instituted, but loculation of the pus and its spread to the opposite side rendered surgical procedures ineffectual.

#### SUBDURAL EMPYEMA AND BRAIN ABSCESS

Case 2 is similar to Case 1 in that suppurative cellulitis spread upward to the base of the skull, causing so much bone destruction in the region of the foramen ovale that a finger could be introduced through the defect. However, the infective process extended further, penetrating the leptomeninges, and the temporal lobe where multiple abscesses formed.

In Case 3 an altogether different pathway was followed to the brain. The floor of the maxillary sinus was fractured during tooth extraction, the ensuing cellulitis spreading into the back of the orbit and then to the frontal bone and frontal sinus. In the region of the more anterior part of the superior orbital plate the orbital infection gained the subdural space and subsequently the base of the frontal lobe.

CASE 2 (AMM Accession 92744).—Male, white, age 43. *Cellulitis of the upper jaw, right, with development of preauricular, zygomaticotemporal and pterygoid abscesses, right; osteomyelitis of the greater wing of the right sphenoid; subdural empyema of the middle cranial fossae; gasserian ganglionitis, right; brain abscesses (temporal lobe), right; acute leptomeningitis; bronchopneumonia with beginning pulmonary abscesses.* Death occurred 28 days after tooth extraction.

*Clinical Data.*—On 7 November, 1942, because of periapical abscesses, the upper right first, second, and third molars (R-6, 7, 8) and the lower right third molar (R-16) were extracted. Local procaine anesthesia was employed. The patient claimed to have been subject to maxillary sinusitis for years. He had been a heavy drinker. Syphilis, contracted ten years previously, was asymptomatic.

Two days after the extractions the right side of the face became painful and swollen and the right eye tender. It was apparent that the cellulitis had its origin in the region of alveoli R-6, 7 and 8. When the patient was admitted to hospital on 14 November, the temperature was 98° F., the pulse 88. There was a moderate discharge from the empty sockets and considerable swelling of the adjacent gum and face. Adjoining cervical lymph nodes were somewhat enlarged.

The gum bordering the empty sockets of the maxilla was incised at once in an effort to promote more adequate drainage; however, on the next day the temperature rose to 102.4° F., the pulse to 92. On 19 November there was a hint of weakness of the left side of the face, and an exaggeration of the left knee jerk. On the following day a ptosis of the right eyelid became apparent.

On 22 November stupor set in. Incision of the right cheek near the site of tooth extraction yielded considerable pus. Examination disclosed inequality of pupils (the right being the larger), ptosis of the right eyelid, edema of the conjunctivae of both eyes, especially the right, engorgement of veins in the fundi but no papilledema, left facial weakness of the central type, and exaggeration of the deep reflexes on the right side with bilateral loss of the abdominal reflexes. There was no proptosis. On 23 November an abscess of the right pterygoid fossa was incised and drained. Cavernous sinus thrombosis being suspected, intravenous treatment with heparin was begun, the dose averaging 180 mg. daily.

On 26 November the patient sank into coma. The swelling of the face had subsided but the edema of the conjunctivae, especially of the right eye, remained. The incised tissues of the right jaw had become necrotic. From the time of admission to 26 November the patient received sulfathiazole therapy, the highest blood level attained being 4.5 mg. per cent. On 27 November sulfadiazine was substituted; the subsequent blood levels of this drug averaged 14.0 mg. per cent. On 28 November, stiffness of the neck and bilateral papilledema became evident. Death occurred 5 December, 28 days after tooth extraction.

*Laboratory Data.*—Blood examinations: on 16 November, RBC 4,300,000; on 19 November, WBC 11,200 (P 93, L 7); on 21 November, WBC 12,900 (P 72, L 27, M 1); on 26 November, WBC 23,550 (P 73, L 23, M 4); on 30 November, RBC 3,930,000, WBC 15,500 (P 66, L 34). Blood culture on 18 November yielded nonhemolytic *Staphylococcus albus*, probably a contaminant. Spinal fluid study on 22 November revealed no changes, but on 30 November showed the following: WBC 650 (P 74, L 36); sugar, 33 mg. per cent; culture, negative. Cultures of pus removed from alveolar abscess on 22 November and from the temporal abscess on 27 November yielded *Staphylococcus aureus*.

*Pathology.*—On removal of the brain, subdural empyema was found to occupy most of the floor of the middle cranial fossa bilaterally; it was particularly abundant on the right side in the region of the sella turcica and the adjacent wall of the cavernous sinus. Sections revealed abscess formation in



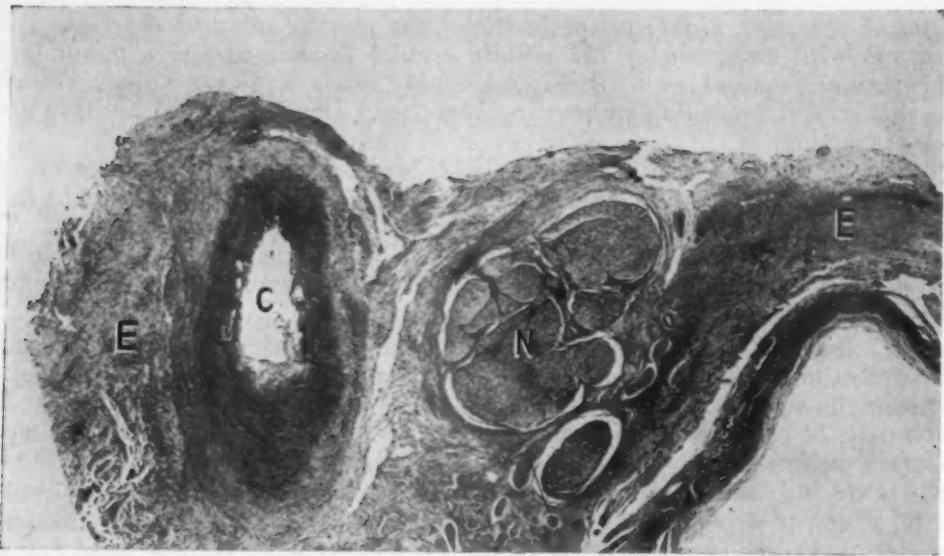


Fig. 3.—Suppuration in the region of the internal carotid and adjacent nerve trunks, right. Considerable purulent exudate (*E*) surrounds the internal carotid (*C*) and the adjacent nerve trunks (*N*). AMM Neg. 83737. Hematoxylin and eosin stain.  $\times 15$ .

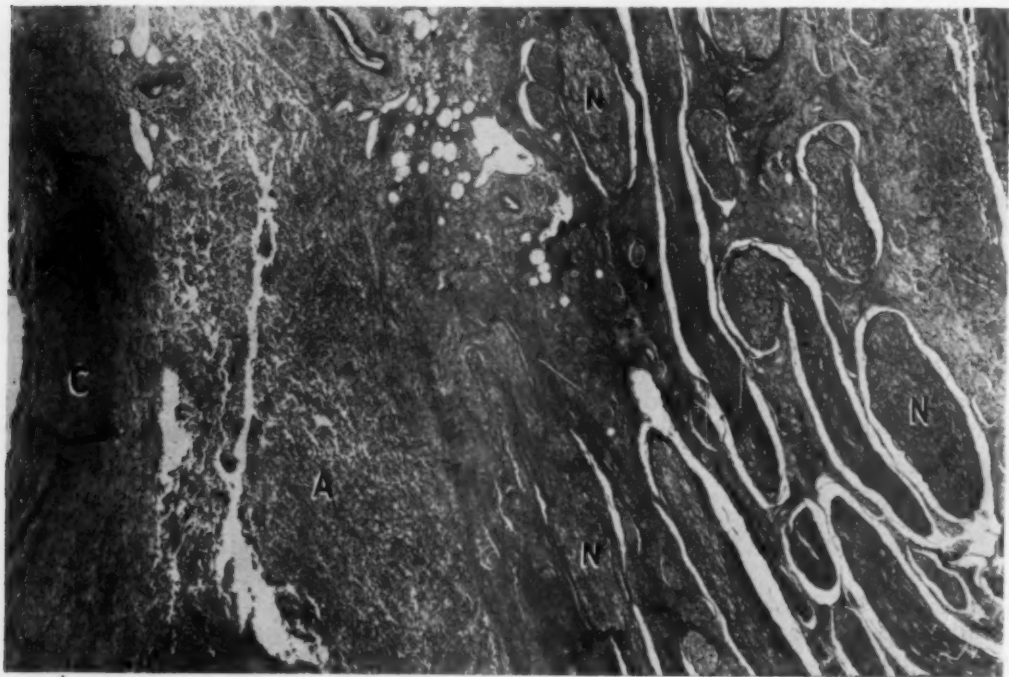


Fig. 4.—Pericarotid abscess, right. The abscess (*A*) is situated between the internal carotid (*C*) and nerve trunks (*N*). A few inflammatory cells are present in the interneural dura. The spaces around the nerve trunks are artifacts. AMM Neg. 83738. Hematoxylin and eosin stain.  $\times 40$ .



the vicinity of the carotid and the oculomotor and trigeminal nerve trunks (Figs. 3 and 4). The cavernous sinuses were empty but the adjacent tissues were heavily invaded by inflammatory cells (Fig. 5). The other venous sinuses showed no change.

Examination of the skull revealed on the right side, just anterior to the foramen ovale, a defect of the middle fossa sufficiently large to admit the little finger. A probe was passed with ease into the necrotic tissues of the pterygoid fossa. The orbits were without gross change but on microscopic examination the subdural space surrounding the right optic nerve was found to contain scanty purulent exudate. No data on the condition of the paranasal sinuses are available.

The leptomeninges at the base of the brain were the seat of moderate purulent exudate. Section of the brain revealed three well-encapsulated abscesses in the right temporal lobe which did not communicate with one another (Fig. 6). Suppurative encephalitis extended to the wall of the adjacent lateral ventricle.

Except for bronchopneumonia and beginning pulmonary abscesses, no other changes were encountered.

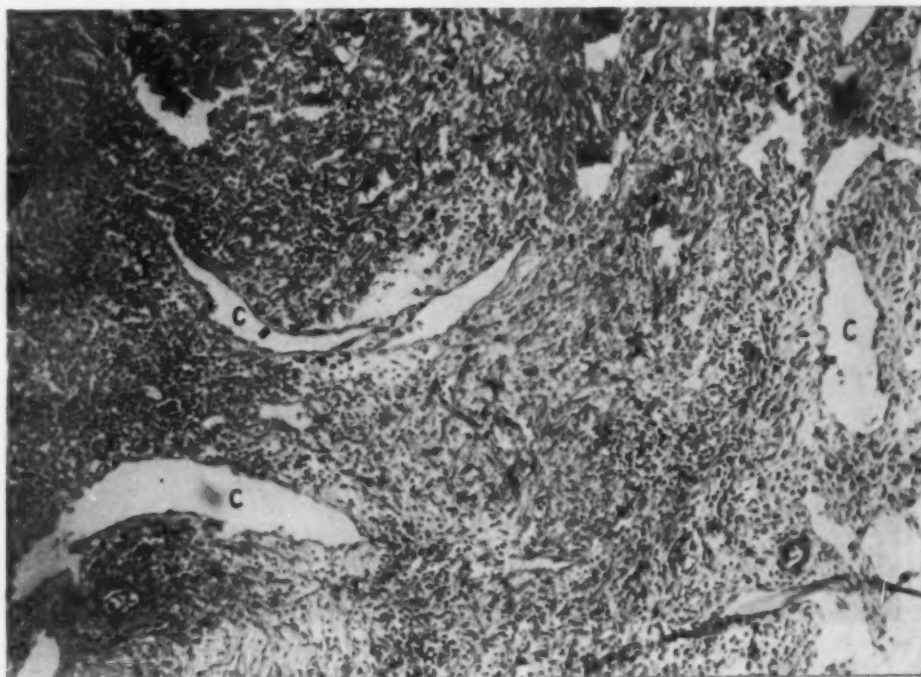


Fig. 5.—Interstitial cavernous sinusitis, right. The channels of cavernous sinus (C) are empty and their endothelium intact, but the interstitial tissue is heavily invaded by inflammatory cells, mostly lymphocytes. AMM Neg. 78735. Hematoxylin and eosin stain.  $\times 125$ .

*Comment.*—The tenderness of the right eye occurring two days after the extraction suggests early extension of the infective process to the region of the intracranial course of the trigeminal nerve. The subsequent conjunctival edema, engorgement of veins in the fundi, inequality of pupils and ptosis, ascribed erroneously to cavernous sinus thrombosis, were due apparently to extension of the inflammatory process to the tissues surrounding the cavernous sinus. It seems logical to assume that the hint of weakness of the left side

of the face and the exaggeration of the left knee jerk on the twelfth day after tooth extraction were evidence of involvement of pyramidal fibers in the region bordering the newly forming temporal lobe abscess.

The abscess in the pterygoid fossa was recognized on the sixteenth day after extraction and duly incised but by that time the intracranial lesions had become well established. The leptomeningitis was terminal.

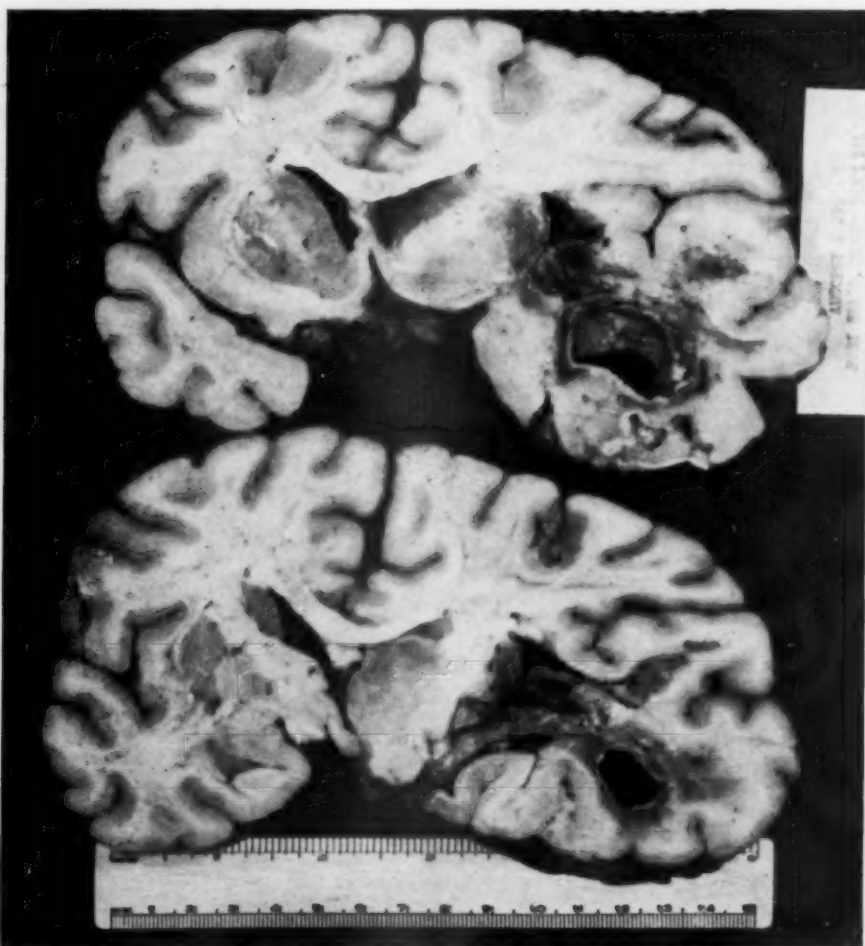


Fig. 6.—Multiple abscesses of the temporal lobe following direct extension of the infective agent through the greater wing of the sphenoid, right. The abscesses are well encapsulated. Inferiorly a sinuous purulent tract reaches the surface of the brain, while medially there is suppurative encephalitis extending to the body of the lateral ventricle. AMM Neg. 83113.

**CASE 3 (AMM Accession 111544).**—Male, white, age 20. *Fracture of the floor of the left maxillary sinus during tooth extraction, with subsequent maxillary sinusitis and osteomyelitis; intraorbital abscess, left; frontal sinusitis and osteomyelitis, left; subdural empyema, left; brain abscess (frontal lobe), left.* Death occurred 129 days after tooth extraction.

**Clinical Data.**—On 12 November, 1943, the following teeth were removed from the upper left jaw; cuspid, first and second bicuspid, and the first, second, and third molars (L-3, 4, 5, 6, 7, 8). The reason for extraction and the type of anesthesia employed are not known. Shortly after the extraction, the region of the left maxillary sinus became painful, and the patient stated that with respirations he could feel air going in and out of the cheek bone.

The patient was admitted to hospital on 12 December. He was febrile and appeared toxic. Bulging of the left eye, which had become apparent two days previously, was the outstanding complaint. Sulfathiazole therapy was begun, the blood level of the drug reaching 6.0 mg. per cent. On 13 December the fluctuant upper lateral portion of the left eyelid was incised and considerable foul-smelling pus released. Blindness of the eye was almost total. By 15 December the left side of the face was swollen and the left pupil failed to react to light or in accommodation; on 16 December there was ill-defined paresis of the right side. The use of sulfathiazole was discontinued and penicillin instituted, a total of 10 million units being given over the succeeding 79 days, 375,000 intrathecally.

On 19 December the first of a series of Jacksonian seizures occurred: the attacks started on the right side of the face and spread to the right arm and right leg. As time went on the patient became irrational and incontinent of urine; the left eyeball became more prominent, the neck stiff, and the Kernig sign positive, and the limbs, both upper and lower, paretic.

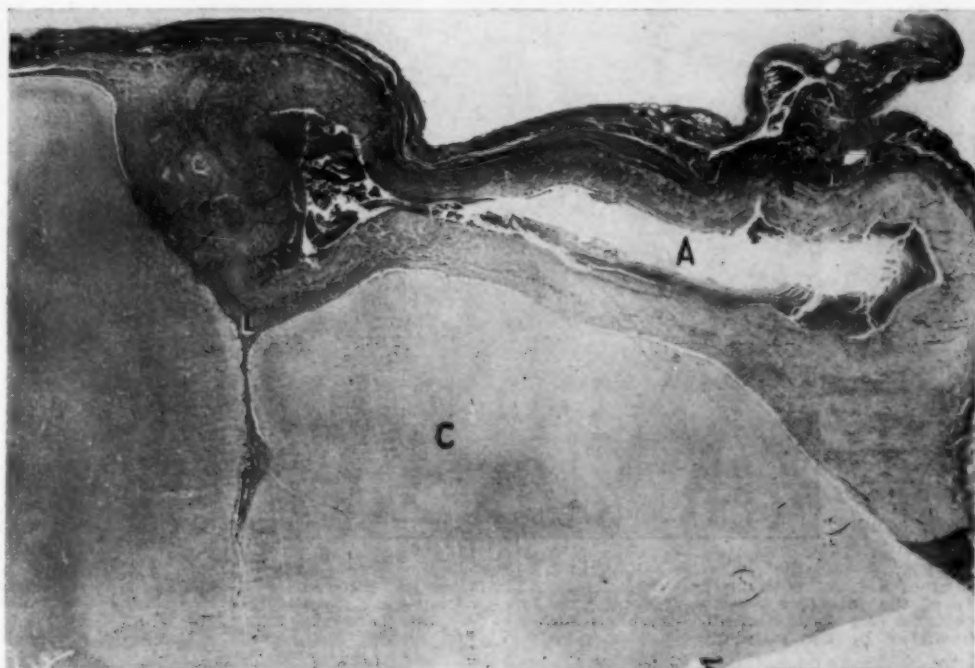


Fig. 7.—Subdural abscess of the mesial surface of the occipital lobe, left. The subdural abscess (A) is well walled off by proliferated connective tissue. The underlying leptomeninges (L), difficult to discern in this photograph, are relatively normal. The cerebral cortex (C) shows no significant change. AMM Neg. 83466. Masson's trichrome stain.  $\times 10$ .

On 4 January, 1944, a radical left frontal sinusotomy was performed. All the walls of the sinus were eroded and purulent but the underlying dura seemed unaffected. After operation the temperature fell to normal, and the patient took a decided turn for the better. On 21 January the left maxillary sinus was drained of pus by the nasal route. One month passed; then because of increasing tenderness of the left cheek and a mounting temperature a left maxillary sinusotomy was performed. A sequestrum,  $3 \times 1 \times 0.5$  cm., was found floating free in the sinus. Shortly thereafter, headaches developed over the left forehead. On 14 March trepanation revealed a large abscess occupying the left frontal lobe of the brain; pus was evacuated, and a rubber catheter inserted to allow further drainage. Death occurred 21 March, 129 days after tooth extraction.



**Laboratory Data.**—Spinal fluid examination on 16 December revealed 24 WBC, and on 19 December, 53 (P 99, L 1); culture yielded hemolytic *Staphylococcus aureus*. Blood examination on 19 December: WBC 16,950. Cultures of pus from left frontal sinus on 4 January showed hemolytic staphylococcus, and from the brain abscess on 14 March hemolytic streptococcus. Blood cultures were repeatedly negative.



Fig. 8.—Mesial aspect of the frontal lobe showing an abscess which is continuous by means of a tract with an epidural abscess. This section illustrates the pathway of extension (P) of the infective process from the epidural region (E) through the dura (D) and leptomeninges (L) to the brain, where an abscess (A) has formed. The point of penetration of the cortex by the purulent tract is clearly visible. AMM Neg. 83465. Mallory's trichrome stain.  $\times 10$ .

**Pathology.**—On exposure of the superior surface of the brain there was no significant change other than adhesion between dura and arachnoid. On removal of the brain, however, a severe and widespread subdural empyema was encountered. Pus coated the entire surface of the left anterior and middle cranial fossae of the left side and extended posteriorly to the pole of the left occipital lobe. In the most anterior part of the left anterior cranial fossa, at the site of surgical removal of the supraorbital ridge, the subdural pus was coextensive with an abscess filling the left orbit. At the point of communication of subdural empyema and intraorbital abscess the dura was friable. In several regions over the base of the skull the empyema had become loculated, forming circumscribed abscesses (Fig. 7); the largest of these,  $4.0 \times 3.5 \times 0.7$  cm. in size, lay between the occipital lobes on the left side of the falx. Leptomeningitis, scanty at best, was present only in the vicinity of subdural suppuration.



On section of the brain, an abscess measuring 3.5 cm. in its greatest diameter was found within the left frontal lobe. It was predominantly basal, extending anteriorly almost to the tip of the frontal pole and as far posteriorly as the level of the optic chiasma; part of it had burrowed into the vicinity of the internal capsule. On the mesial aspect of the hemisphere, a well-delineated tract could be followed from the epidural tissue to the brain abscess (Fig. 8); the tract consisted of a matrix occupied by proliferated blood vessels and many lymphocytes, plasma cells, and polymorphonuclear leucocytes. The brain abscess reached the wall of the anterior horn of the lateral ventricle but the ependyma remained intact.

The lining of the left maxillary sinus was thickened and nodular. The sphenoidal, ethmoidal, and right maxillary sinuses as well as the middle ears were normal. The same was true of the intracranial venous sinuses.

Examination of the thoracic and abdominal viscera revealed no abnormalities.

*Comment.*—On admission to hospital one month after tooth extraction the diagnosis of orbital abscess was obvious. Incision and drainage of the orbit did not, however, halt the process, for on the thirty-seventh day after extraction there was evidence of intracranial extension, as manifested by Jacksonian seizures, mental disturbances, and incontinence of urine. The prolongation of the subsequent course may be attributed to the therapeutic effects of penicillin.

#### LEPTOMENINGITIS

Leptomeningitis was a terminal complication in numerous cases of this series, but in two instances it proved to be the essential intracranial lesion. In the first of these (Case 4) the infection was hematogenous; the leptomeningitis occurred early in the course of the illness and would doubtless have become florid had not the bacteremia been so rapidly fatal. In the other (Case 5), of longer duration, the leptomeningitis was the consequence of direct spread of the infective process through the base of the skull; local suppurative pachymeningitis marked the site through which the infection had passed.

**CASE 4 (AMM Accession 36331).**—Male, white, age 23. *Cellulitis at the angle of the left jaw and the upper cervical region; edema of the floor of the mouth, epiglottis and larynx; abscess of the submaxillary and upper cervical regions, left; bacteremia; early leptomeningitis.* Death occurred 5 days after tooth extraction.

*Clinical Data.*—On 2 September, 1931, the lower left third molar (L-16) was extracted. Neither the reason for extraction nor the type of anesthesia employed are known. The other teeth were in good condition and the hygienic condition of the mouth was normal. There had been no significant previous illnesses.

On 6 September the patient was admitted to hospital because of painful swelling of the jaw and neck of the left side. The temperature was 102.6° F., the pulse 118. The tissues beneath the angle of the jaw were swollen and fluctuant, and the adjoining submental region and the floor of the mouth were edematous. The regional lymph nodes were considerably enlarged. Trismus was severe.

On the day of admission, under procaine anesthesia, one incision was made beneath the angle of the left jaw, and another in the submental region. Some 20 c.c. of foul-smelling pus escaped at the site of the first incision. The patient seemed better, but on 7 September, thirteen hours after operation and five days after tooth extraction, he was found dead.

**Laboratory Data.**—Blood examination on 6 September: WBC 23,800 (P 90, L 7, M 2, B 1). Spinal fluid taken by cisternal puncture post mortem contained 95 cells per c.mm. Cultures of heart blood and of pus from the cervical region yielded a heavy growth of hemolytic streptococci.

**Pathology.**—Dissection of the region of the operative incisions revealed a layer of pus surrounding the sternomastoid and sternohyoid muscles. The inflammatory process did not extend into the mediastinum.

Examination of the laryngeal region disclosed considerable edema of the epiglottis, the aryepiglottic folds, and the false vocal cords. The lungs and pleurae contained numerous petechiae. Within the fissure between the upper and middle lobes of the right lung a moderate-sized hemorrhage was found.

The leptomeninges were extremely hyperemic, and along the course of the larger vessels they were cloudy. No thrombi were observed. Section of the brain showed nothing significant.

**Comment.**—This case was remarkable for the rapid spread of the suppurative process. Death was due to an overwhelming bacteremia, the effects of which were more evident in the lungs and leptomeninges than elsewhere.

**CASE 5 (AMM Accession 29149).**—Male, white, age 52. *Fracture of the floor of the right maxillary sinus during tooth extraction; abscesses, preauricular, subtemporal, and submandibular, right; osteomyelitis of the maxilla and the greater wing of sphenoid, right; maxillary and sphenoidal sinusitis, right; purulent pachymeningitis of the middle cranial fossa, right; acute leptomeningitis; thrombosis of the right ophthalmic artery, with rupture into the leptomeninges.* Death occurred 27 days after tooth extraction.

**Clinical Data.**—On 5 March, 1928, the upper right third molar (R-8) was extracted. The tooth had ached intermittently for six months. No data on preliminary roentgenography or of anesthesia employed are at hand. At an unstated time after extraction the patient began to suffer from pain in the right upper jaw. Swelling of the right side of the face became apparent, and a slight headache developed.

When admitted to hospital on 17 March the patient's temperature was 102.6° F., the pulse 98. Diffuse redness and swelling of the entire right side of the face included the right eyelids. The submandibular lymph nodes were considerably enlarged. Pus could be expressed from most of the tooth margins. By 19 March the swelling had spread to the soft palate and pharynx, and by the next day there was fluctuation on the right side in the region of the temple, the preauricular region, and the angle of the jaw. Incisions at these sites released copious pus. A small abscess of the gum over the upper right lateral incisor (R-2) was also drained.

On 20 March the patient had a convulsive seizure. Up to that time the temperature had fluctuated between 99.8 and 103.2° F., the pulse from 72 to 94. On 27 March a distinct paresis of the left upper limb and the left side of the face was evident; the left pupil was found to be larger than the right and remained so. The ocular fundi were normal.

On 31 March the neck became rigid, opisthotonos developed, unconsciousness supervened, and death occurred 1 April, 27 days after tooth extraction.

**Laboratory Data.**—Blood examination on 18 March: WBC 14,950 (P 85, L 15), and on 24 March, WBC 14,800 (P 88, L 12). Culture of pus from the right preauricular abscess on 20 March revealed hemolytic staphylococci; smears showed Vincent's organisms. Blood cultures on 20 and 22 March were negative but culture of heart blood at autopsy yielded hemolytic streptococci.

**Pathology.**—At autopsy a probe could be passed through the socket of the removed tooth into the maxillary sinus where considerable pus was encoun-

tered. In the parietal and temporal regions the scalp was undermined by pus. The fascia over the temporal muscle had sloughed away and neighboring muscles were necrotic.

On examination of the skull, with brain removed, the greater wing of the right sphenoid (near the foramen ovale) was found to be necrotic and the overlying dura purulent. Bone destruction had progressed until the right maxillary and right sphenoidal sinuses intercommunicated. When pressure was exerted on the tissues in the right temporal region, pus welled up through the eroded sphenoid into the right middle cranial fossa. The leptomeninges, including those of the posterior cranial fossa and spinal canal, were purulent and hemorrhagic. Dissection revealed thrombosis of the right ophthalmic artery and the site where its rupture had led to the leptomeningeal hemorrhage. Section of the brain revealed nothing of import.

*Comment.*—Appearances at autopsy indicated that during extraction of the third molar the maxillary sinus was broken open. Maxillary sinusitis followed, and in the meantime the infective process spread to the greater wing of the sphenoid and to the sphenoidal sinus. On the fourteenth day after extraction, the abscess in the temporal region was incised and drained, but by that time the infection had penetrated the skull, as manifested on the fifteenth day by a convulsive seizure.

#### LEPTOMENINGITIS AND BRAIN ABSCESS

Leptomeningitis may, for a time, be the outstanding manifestation of intracranial extension, and may in fact be brought under control by antibiotic drugs so that cure is in sight, when, suddenly, stupor sets in and death supervenes, the cause of which is found at autopsy to be brain abscess. This was the turn of events in two cases of the series. In each the leptomeningitis and brain abscess arose concurrently, only the meningeal infection responding to therapy. The first case was one of direct spread of the infection through the skull (Case 6), while the second was by way of the general circulation (Case 7).

**CASE 6** (AMM Accession 108469).—Male, white, age 28. *Cellulitis of the upper jaw, right; abscesses, alveolar and subtemporal, right; osteomyelitis of the greater wing of the sphenoid and the squamous portion of the temporal bone, right; sphenoidal sinusitis; purulent pachymeningitis of the middle cranial fossa, right; leptomeningitis which responded to sulfadiazine; brain abscess (temporal lobe), right; acute subarachnoid hemorrhage; pons; acute bronchopneumonia.* Death occurred 38 days after tooth extraction.

*Clinical Data.*—On 25 February, 1944, because of periapical abscesses, the upper and lower right third molars (R-8, R-16) were removed, and on 14 March the lower left third molar (L-16). Both extractions were done under conduction anesthesia. The socket of the upper right third molar (R-8) was the site of the subsequent trouble.

The patient entered hospital 16 March with painful swelling of the right side of the face, especially at the angle of the jaw. The temperature was 99.8° F., the pulse 92. The diagnosis of acute nonsuppurative osteitis was made clinically. Three days later the patient had a chill, and the temperature mounted to 103.8° F. On 20 March he became disoriented and semicomatose; vomiting occurred, a severe headache developed, and the neck became stiff. A spinal tap revealed meningitis. Sulfadiazine was given, the subsequent blood level of the drug averaging 11.0 mg. per cent.

On 22 March, because of fluctuant swelling, the right subtemporal fossa was incised, and 100 c.c. of pus evacuated; on the next day an alveolar abscess



in the region of R-8 was incised and drained intraorally. On 24 March the patient became drowsy. His pulse averaged 64. Penicillin therapy was instituted, the total dose over four days being 415,000 units. The patient's general condition improved and the facial swelling almost disappeared; however, on 29 March an intense and intractable frontal headache developed.

On 1 April the patient became stuporous. The tendon reflexes of the upper and lower limbs of the right side were found to be hyperactive. The fundi were not altered. Death occurred 3 April, 38 days after tooth extraction.

**Laboratory Data.**—Blood examinations: on 21 March, WBC 14,500 (P 84, L 13, M 2, E 1); on 22 March, WBC 13,400 (P 76, L 22, M 2); on 25 March, WBC 10,750 (P 60, L 34, M 4, E 1, B 1). Spinal fluid examination: WBC on 20 March, 11,000 (P 92, L 8); on 22 March, 400 (P 97, L 3); on 25 March, 41 (P 92, L 8). Cultures of spinal fluid on three occasions were negative; of blood on 20 and 31 March, no growth; of sputum on 24 March, hemolytic *Staphylococcus aureus* and nonhemolytic streptococci; of subtemporal abscess on 24 March, anaerobic *Staphylococcus aureus*; of brain abscess post mortem, facultative anaerobic staphylococci and streptococci, types unspecified.

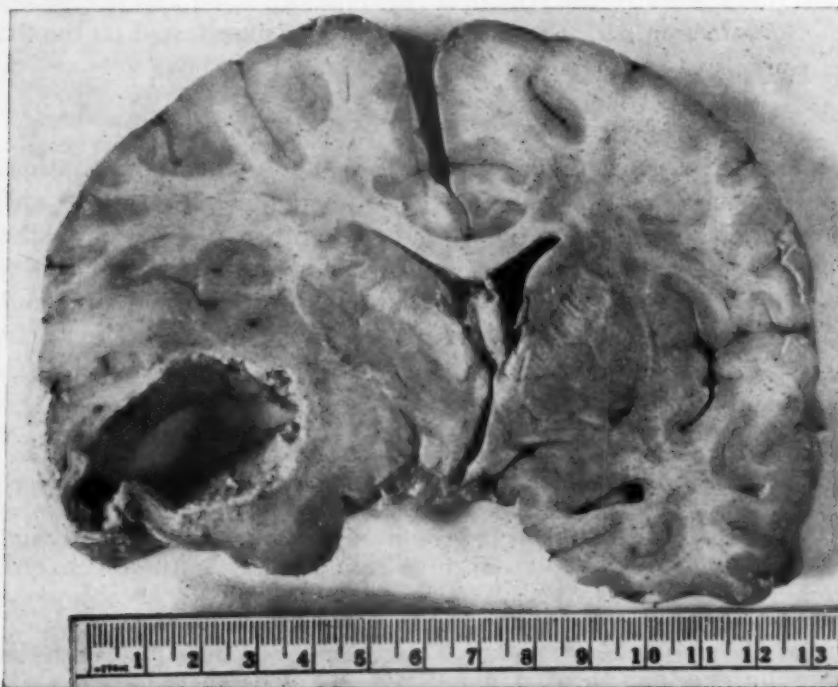


Fig. 9.—Abscess of the right temporal lobe resulting from upward extension of infection through the greater wing of the sphenoid bone. The meninges adjacent to the abscess are greatly thickened. Note the distortion of the lateral and third ventricles. AMM Neg. 80299.

**Pathology.**—Other than bronchopneumonia the viscera showed nothing of import. On removing the brain, an adhesion between the dura and the inferior aspect of the right temporal lobe was encountered. When an attempt was made to break the adhesion a copious amount of pus escaped from the temporal lobe. In the region of the adhesion the inner surface of the dura was coated with an exudate, 3 cm. in diameter. There was erosion of the middle cranial fossa, especially in the region just lateral to the foramen ovale. The squamous portion of the temporal bone was similarly involved. A probe could be readily passed through the sphenoid bone down to the inner surface of the masseter, where creamy pus was encountered. The sphenoidal sinus was filled



with pus, but other paranasal sinuses showed nothing of note. The dural venous sinuses were normal.

Section of the brain revealed an abscess of the right temporal pole,  $4 \times 3 \times 2$  cm. in size (Fig. 9). A subarachnoid hemorrhage of moderate degree was present around the base of the pons.

*Comment.*—Leptomeningitis became evident on the twenty-fourth day after tooth extraction and, as judged by the spinal fluid studies, had virtually subsided by the twenty-ninth day. Subsequent symptoms are attributable to the brain abscess, and the final event to sudden subarachnoid hemorrhage from vessels in the vicinity of the pons.

**CASE 7** (AMM Accession 96020).—Male, white, age 24. *Presumed bacteremia at the time of extraction; leptomeningitis which responded to sulfadiazine; brain abscess (temporal lobe), right.* Death occurred 41 days after tooth extraction.

*Clinical Data.*—When first seen in the dental clinic 6 January, 1943, the patient had Vincent's stomatitis. After two weeks of treatment the infection was brought under control. Subsequently, several teeth were treated for caries. On 8 February an impacted lower right third molar (R-16) was extracted. This passed without incident. Then on 28 April, because of roentgenologic evidence of periapical abscess (Fig. 10), the upper left first bicuspid (L-4) was removed. Local anesthesia was employed.



Fig. 10.—Roentgenogram of periapical abscess. The bone in the region of the upper left first bicuspid (L-4) is considerably rarefied. AMM Neg. 76571.

The following day the patient began to have severe headaches. Two days later the left arm and leg became weak. Owing to increasing severity of headaches the patient was admitted to hospital on 6 May. The temperature was  $102^{\circ}\text{F}$ ., the pulse 92. The gums in the region of extraction were well healed. Hemiplegia of the left side was evident. The neck was stiff, and Kernig's sign was elicited on the left. On 8 May the right pupil was found to be larger than the left. Spinal tap yielded turbid fluid. Sulfadiazine therapy was begun; during the next month the blood level of the drug averaged 12.0 mg. per cent.

Toward the latter part of May the patient's general condition improved, the hemiparesis diminished, and on 1 June he was allowed to sit up in a chair for a short time. The next two days saw continued improvement, but on 4 June the headaches recurred, nausea set in, and complete motor aphasia developed. Stupor ensued. Exploration for suspected brain abscess was per-

formed on 6 June but no lesion was found. On 8 June, 41 days after tooth extraction, the patient died.

**Laboratory Data.**—Spinal fluid examinations: on 3 May, negative; on 8 May, WBC 10,000; on 10 May, 633; on 31 May, 83; on 5 June, 48. Blood examinations: on 6 May, WBC 35,200 (P 95, L 4, M 1); on 7 May, WBC 22,150 (P 97, L 2, M 1); on 12 May, WBC 13,800 (P 81, L 16, M 2, E 1); on 14 May, WBC 11,000 (P 79, L 13, M 5, E 3); on 17 May, WBC 11,600 (P 82, L 17, M 1). Culture of pus from brain abscess post mortem yielded a mixed flora with *Streptococcus viridans* predominating.

**Pathology.**—At autopsy the only changes of note were intracranial. The dural sinuses, the skull, the paranasal sinuses and the petrous portion of the temporal bones were normal. Examination of the leptomeninges disclosed no free exudate, but at the base of the brain and around the brain stem the leptomeninges were thickened. On section of the brain, the right temporal lobe was found to contain a well-encapsulated abscess 3.0 cm. in diameter. It was situated 2.5 cm. beneath the surface of the cortex. Microscopically the leptomeninges of the base of the brain contained moderate numbers of lymphocytes.

**Comment.**—The rapid onset of symptoms after extraction suggests immediate spread to the intracranial cavity by way of the general circulation. Under the influence of sulfadiazine the leptomeningitis had almost disappeared when on the thirty-seventh day the patient became comatose. The brain abscess was on the side opposite the tooth extraction.

#### SUPPURATIVE ENCEPHALITIS AND EPENDYMITIS

Infections traveling to the brain by way of the general circulation may lead to suppurative encephalitis which may then spread to the ventricle, leading to ependymitis and leptomeningitis. Of such a complication there is only one example in this series.

**CASE 8 (AMM Accession 100875).**—Male, white, age 26. *Cellulitis of the mandibular region with osteomyelitis of the mandible, right; early abscesses of the left kidney; suppurative encephalitis, left; purulent ependymitis and supuration of the choroid plexus, left lateral ventricle, with subsequent acute leptomeningitis.* Death occurred 54 days after tooth extraction.

**Clinical Data.**—On 29 April, 1943, under block anesthesia, the lower right third molar (R-16) was extracted. The reason for extraction is not known. Pain in the lower jaw persisted for a few days thereafter.

One month after extraction (1 June) the patient was admitted to hospital because of swelling of the right jaw at the site of extraction. The temperature and the pulse rate were normal. Drainage of alveolus R-16 was established surgically, after which the socket was irrigated with azochloramine solution and dusted with sulfanilamide.

By 4 June the temperature was elevated, and within a few days climbed to 103° F. On 6 June sulfadiazine treatment was begun; subsequent blood levels of the drug averaged 7.0 mg. per cent. On 7 June a sequestrum about the size of a tooth was removed from the involved part of the mandible. The next day a cough developed, and there were roughening of breath sounds and a few fine râles at the base of the left lung. There had been no chills.

On 14 June the patient appeared toxic and complained of generalized soreness of muscles. The anterior cervical lymph nodes were somewhat enlarged. The cellulitis of the jaw had receded and the lungs had cleared, but on 20 June the temperature again began to rise. On the next day the patient became stuporous. Hemiplegia of the right side was apparent, and the neck stiff. Death occurred on 22 June, 54 days after tooth extraction.

*Laboratory Data.*—Blood examinations: on 8 June, RBC 3,980,000, WBC 5,800 (P 61, L 38, M 1); on 10 June, RBC 4,100,000, WBC 6,900 (P 53, L 45, M 2); on 20 June, WBC 14,700 (P 78, L 16, M 6). Roentgenograms on 10 June showed nothing of significance in the mandible surrounding empty socket R-16 (Fig. 11). Blood cultures were negative on three occasions. Spinal fluid on 21 June: WBC 177 (P 41, L 59).



Fig. 11.—Roentgenogram of the right jaw showing the socket previously occupied by the third molar (R-16). The roentgenogram was taken on the forty-second day after extraction. A small dressing is present in the socket. AMM Neg. 77511.

*Pathology.*—At autopsy no gross changes in the viscera were detected, but on microscopic examination the left kidney was found to contain several large accumulations of polymorphonuclear leucocytes and round cells. These were present in both cortex and medulla. Within some of the foci the parenchyma was in a state of necrosis. Other than round cell infiltration of the epicardium and the periadrenal tissue no further changes were noted.

After removal of the brain, nothing of consequence could be found in the skull, the paranasal sinuses, or the venous sinuses. On section of the brain, the ventricles were found to be considerably dilated. In the left lateral ventricle the choroid plexus was matted with yellowish exudate. Within this ventricle, as well as in the third and fourth, there was an abundance of necrotic material. The periventricular tissue was softened and discolored, and in the region of the septum pellucidum the brain was necrotic for a depth of about 2.0 cm. There was no frank brain abscess.

Microscopically the choroid plexus of the left lateral ventricle was heavily overlaid by purulent exudate, and in places was invaded by inflammatory cells (Fig. 12). Perivascular clumps of bacteria were not observed. The subependy-



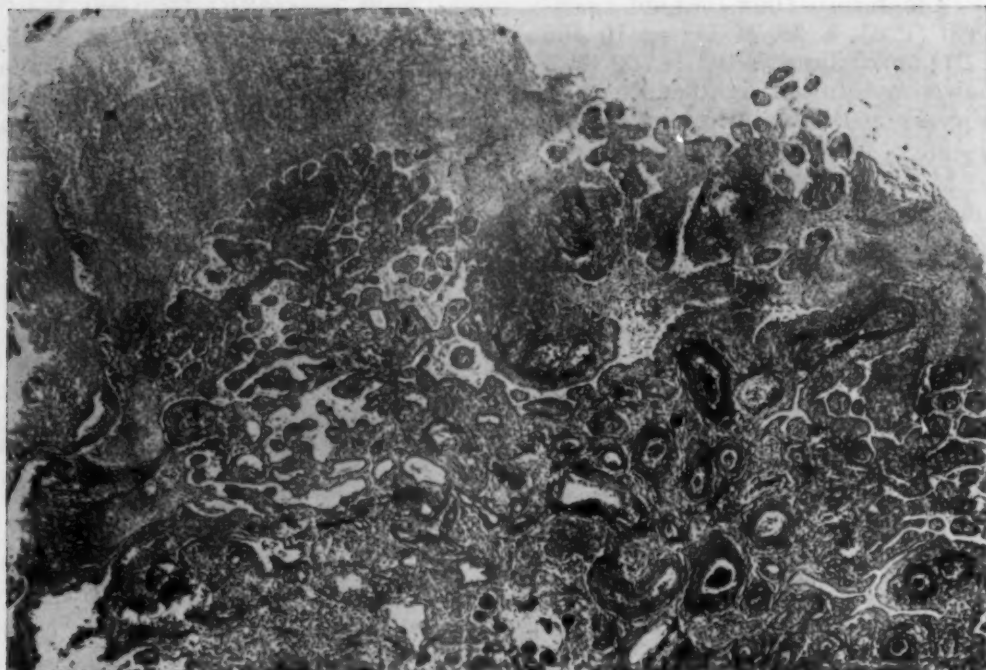


Fig. 12.—Suppuration of the choroid plexus of the left lateral ventricle. The fronds of the choroid plexus are moderately infiltrated with inflammatory cells, and adjacent to them, lying free in the ventricle, is a profuse purulent exudate. AMM Neg. 84505. Hematoxylin and eosin stain.  $\times 30$ .

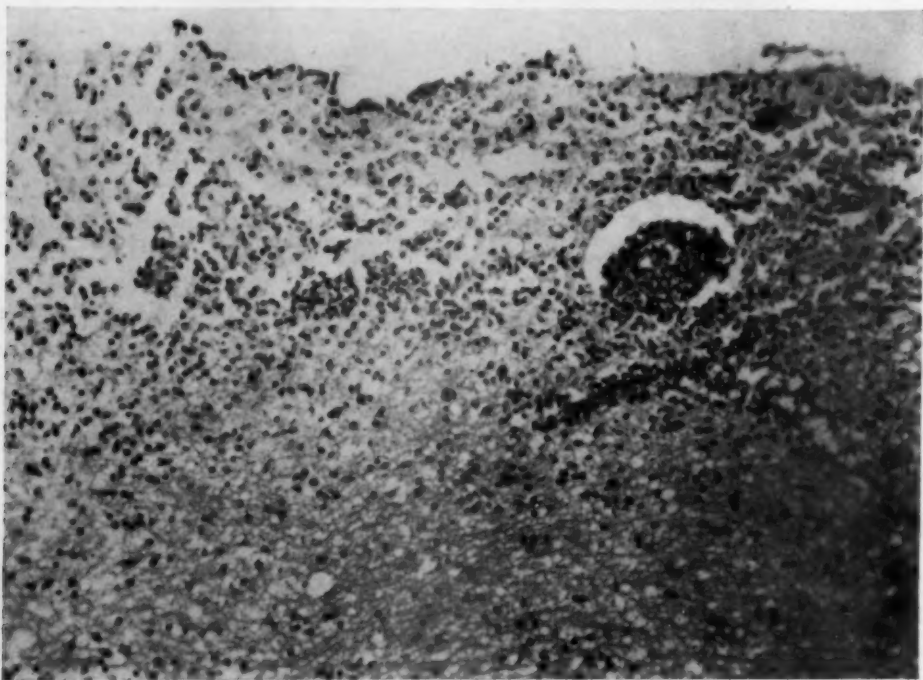


Fig. 13.—Ependymitis of the left lateral ventricle. Part of the ependymal lining is intact. The periventricular system contains many inflammatory cells; the same is true of perivascular spaces. AMM Neg. 78723. Hematoxylin and eosin stain.  $\times 150$ .



mal tissue of the entire ventricle was extensively invaded by inflammatory cells (Fig. 13). Perivascular cuffs of inflammatory cells extended in diminishing degree into the surrounding white matter. No sample of brain in the region of the gross suppurative encephalitis was available for study. Leptomeningitis was present over the base of the brain.

*Comment.*—The sequence of events appears to be as follows: About one month after extraction the previously smoldering infection of the mandible flared up. Roentgenologic study revealed a sequestrum in the affected region. It is quite possible that as a result of operative manipulation in the removal of the sequestrum the infective organism gained the blood stream, and thus was carried to the left kidney and subsequently to the brain in the region of the lateral ventricle. It is probable that brain abscess would have developed had not the infective organism gained entrance into the lateral ventricle at such an early stage.

#### METASTATIC BRAIN ABSCESS

Metastatic, or hematogenous, brain abscess not preceded by leptomeningitis or pachymeningitis was encountered in five cases of this series. They fall into two groups: (1) those characterized by a single large abscess in the frontoparietal region of the brain and by the absence of metastatic abscesses elsewhere in the body (Case 9, 10, and 11), and (2) those in which brain abscesses were multiple and in which the infection was disseminated also in thoracic or abdominal organs, or both (Cases 12 and 13).

**CASE 9 (AMM Accession 94633).**—Male, white, age 32. *Presumed bacteremia occurring at the time of extraction; brain abscess (frontoparietal), right; acute leptomeningitis; acute bronchopneumonia.* Death occurred 23 days after tooth extraction.

*Clinical Data.*—The patient was admitted to hospital 30 March, 1943, for investigation because of pains and swelling of the knees and hips which had occurred at intervals during the preceding year. He also had been subject to frequent headaches centering somewhat above both ears; these occurred in the morning and disappeared as the day wore on. Going on long hikes had accentuated the symptoms. The patient appeared well. No tangible evidence of arthritis could be found. Various laboratory tests proved negative. There was considerable caries of five teeth, and roentgenologic examination disclosed a periapical abscess of the upper left bicuspid (L-5). On 23 April, under local procaine anesthesia, the bicuspid was removed.

After the extraction there was no undue local swelling of the gums or of lymph nodes. Days passed without incident, when on 3 May the patient had a convulsive seizure followed in short succession by nine more. Up to this time the temperature had been in the normal range, except on 27 April when it reached 99.4° F. The pulse, however, had risen somewhat since the extraction, on three occasions ranging between 88 and 100.

The day after the convulsive seizures, recurrent intense headaches set in. Ophthalmic examination revealed no abnormalities. On 8 May nausea and vomiting began, and the headache became intractable. On 15 May the neck became rigid. The right pupil was dilated, the left constricted. The margins of the optic discs were hazy, the fundal vessels were engorged, and there were several retinal hemorrhages. Babinski and Oppenheim signs were elicited bilaterally. The patient lapsed into coma. Sulfadiazine therapy was instituted, the blood level of the drug reaching 17.0 mg. per cent. A needle was

introduced through burr holes made in the right parietal region and pus evacuated from a brain abscess. Death occurred 16 May, 23 days after tooth extraction.



Fig. 14.—Roentgenogram of jaw taken on the twenty-third day after extraction of the upper left second bicuspid (L-5). Periapical rarefaction is present in the region of the extracted tooth. AMM Neg. 80245.

*Laboratory Data.*—Blood examination on 15 May: WBC 19,500 (P 80, L 20). A roentgenogram on 16 May showed periapical rarefaction of bone in the region of the extracted upper second premolar (Fig. 14). Spinal fluid examination on 15 May: WBC 1,250 (P 90, L 10); sugar, 125 mg. per cent; colloidal gold curve, 4332100000. Roentgenograms of knees and cervical spine on 24 and 27 April revealed minimal arthritic change. Culture of spinal fluid on 15 June, no growth; of pus from brain abscess, streptococci, type unspecified.

*Pathology.*—Other than early bronchopneumonia, the thoracic and abdominal viscera were without change. At the site of the tooth extraction there was no evidence of cellulitis. After removal of the brain no abnormalities could be found in venous sinuses, paranasal sinuses, or petrous part of the temporal bones. Examination of the removed brain revealed leptomeningeal exudate of moderate amount, especially at the base of the brain. Section of the brain disclosed an abscess, 4.0 cm. in diameter, occupying the posterior part of the right frontal lobe and the adjoining parietal lobe. The abscess cavity was lined by a grayish membrane 2.0 mm. in thickness. Parenchyma adjacent to the wall of the abscess was found on microscopic examination to be heavily invaded by inflammatory cells.

*Comment.*—In this instance the pulse rate increased on the second day and there was transient fever on the third day after the tooth extraction. Except for continued elevation of the pulse the course was uneventful until ten days after extraction when the patient had a series of convulsive seizures, which may be ascribed to changes incident to early formation of the brain abscess. Then signs of bilateral pyramidal tract disturbance became manifest. Twelve days after onset of the convulsive seizures the infective process spread to the leptomeninges.

Since the brain abscess developed in the right hemisphere, whereas the tooth was extracted from the left maxilla, and since there is no evidence to support a direct spread, it seems logical to conclude that transient bacteremia occurred at the time of tooth extraction and that the organisms settled only in the brain.

CASE 10 (AMM Accession 90134).—Male, white, age 31. *Presumed bacteremia occurring at the time of extraction; brain abscess (frontoparietal), left; acute leptomeningitis, left.* Death occurred 137 days after tooth extraction.

*Clinical Data.*—On 24 September, 1942, owing to malposition, the upper left and upper right lateral incisors (L-2, R-2) were removed under local infiltration anesthesia, and replaced by a temporary denture. After a few days a partial denture was substituted; on 17 November the prosthesis was completed. Roentgenograms taken two days prior to extraction showed nothing beyond malposition.

The patient was well for about four months when, on 31 January, 1943, he developed weakness and numbness of the right arm and hand. At work that day he noted that his handwriting had deteriorated. Headaches began. The next morning he was able to lace his shoes only after repeated efforts. Later he experienced some difficulty in walking and was unable to turn his eyes completely to the right.

When admitted to hospital, 4 February, the patient was acutely ill, agitated, and somewhat disoriented. There was slurring of speech, but aphasia was not apparent. The temperature was 99° F., the pulse 68. The pupils were regular and reacted to light and in accommodation. The visual fields were full and the optic discs showed no change. There was a right internal strabismus with moderate limitation of lateral motion of the eyes. The lower two-thirds of the right side of the face was paretic, the arm and leg of the right side weak and slightly spastic. Examination of sensibility disclosed on the corresponding side a hypesthesia, a loss of appreciation of position of the digits, and astereognosis.

On 5 February there were two episodes of projectile vomiting. The temperature rose to 99.4° F., the pulse to 78. Progression was evident: the right side of the face had become more paretic and hypesthetic, and there was conjugate deviation of the eyes to the left with inability to turn them to the right. Death occurred on 8 February, 137 days after tooth extraction.



**Laboratory Findings.**—Blood examination on 5 February: RBC 4,980,000; WBC 7,300 (P 74, L 26). Spinal fluid examination on 6 February, WBC 7 (all lymphocytes); total protein, 75 mg. per cent; chlorides 528 mg. per cent; gold curve 0000000000; sugar, 105 mg. per cent; Kahn, negative. Blood culture on 5 February, no growth. Culture of pus from brain abscess yielded anaerobic *Staphylococcus aureus*.

**Pathology.**—At autopsy the only changes of significance were intracranial. Over the convex surface of the left cerebral hemisphere, especially in the region of the lateral fissure, the leptomeninges contained a slight purulent exudate. Section of the brain revealed a well-encapsulated abscess, 3 cm. in diameter, situated in the white matter of the posterior frontal and anterior parietal lobes. Exploration of the temporal bones, the venous sinuses, and the paranasal sinuses revealed no abnormalities. On microscopic examination, the abscess wall was found to be of considerable thickness and beyond it there was a pronounced astrocytosis which extended to the surface of the cortex. The overlying meninges contained focal accumulations of lymphocytes, histiocytes, and a few polymorphonuclear leucocytes.

**Comment.**—The histologic appearance of the wall of the brain abscess is indicative of chronicity. Since at autopsy no focus of pyogenic infection could be found, it seems reasonable to assume that the brain abscess was a complication of the tooth extraction and that the infection was blood-borne. That brain abscess may, in fact, lie dormant for this length of time is well authenticated (Wartenberg, Nauwerck). A similar case, in which brain abscess became manifest five months after otherwise uncomplicated tooth extraction, has been described by Parker.

**CASE 11 (AMM Accession 45341).**—Male, white, age 38. *Local osteomyelitis and cellulitis of the mandibular region, left; presumed bacteremia occurring at time of extraction of the lower left second molar; brain abscess (frontoparietal), right; acute bronchopneumonia.* Death occurred 49 days after tooth extraction.

**Clinical Data.**—On 16 November, 1934, because of impaction, the lower left third molar (L-16) was extracted. The type of anesthetic and its mode of administration are not known. For a few days subsequent to extraction there was painful swelling of the postmandibular region.

The patient was admitted to hospital on 10 December. The temperature was 98° F., the pulse 80. The left side of the face, especially in the region of the angle of the jaw, was considerably swollen. The lower left second molar (L-15) was extracted in an effort to drain the region. The patient's general condition was improving when, on 23 December, he had a generalized convulsive seizure, followed later in the day by three more. The next day there were bradycardia and vomiting as well as mental confusion and somnolence. A low-grade fever ensued but it soon subsided. On 28 December and again three days later, small sequestra were removed from the region of L-15. Vomiting continued with increased frequency. Neurologic examination, including repeated observation of the ocular fundi and the pupils, failed to reveal evidence of organic disease.

On 3 January another series of convulsive seizures occurred. Death ensued 4 January, 49 days after tooth extraction.

**Laboratory Data.**—Blood examinations: on 23 December, WBC 24,000 (P 96, L 4); 3 January, WBC 11,000 (P 71, L 29); on 4 January, WBC 27,000 (P 85, L 15). Blood chemistry on 24 December: sugar 99, urea n. 25.4 (later 27.2, 18.0, 26.3, 18.7), CO<sub>2</sub> combining power 50.4, calcium 9.8 mg. per cent. Spinal fluid examinations on 24 and 31 December were normal; on 3 January, RBC 71, WBC 22. Cultures were negative. Roentgenograms of mandible (date



not given) showed evidence of osteomyelitis in regions L-15 and L-16; on the right the antrum was opaque. Post-mortem cultures of ethmoidal and sphenoidal sinuses and of the brain abscess yielded pneumococcus (which was not specifically typed) and *Staphylococcus aureus*; and of the right middle ear, *Staphylococcus aureus*.

*Pathology.*—Examination of the skull after removal of the brain revealed a considerable amount of tenacious mucoid material in the sphenoidal sinuses and in the right middle ear. The frontal and ethmoidal sinuses were normal; the maxillary sinuses were not explored.

The leptomeninges were of normal appearance. Section of the brain revealed an abscess, 5 cm. in diameter, in the right frontal and adjoining parietal lobe; it was lined by a well-formed pyogenic membrane surrounded by a narrow zone of capillary hemorrhage. The lungs showed early bronchopneumonia.

*Comment.*—From the lack of evidence of direct spread of the mandibular osteomyelitis and cellulitis following extraction, and from the fact that the brain abscess was on the side opposite the site of tooth extraction, one may conclude that the brain abscess was hematogenous. The bacteremia probably occurred at the time of extraction of the lower left second molar (L-15). The presence of the same organism (pneumococcus) in the brain abscess and in the ethmoidal and sphenoidal sinuses appears to be coincidental.

CASE 12 (AMM Accession 95901).—Male, white, age 26. *Cellulitis of the left mandibular and adjoining cervical region; submaxillary abscess, left; multiple pulmonary abscesses, bilateral, with pleural empyema, right; brain abscesses, all lobes; thrombophlebitis of the cavernous sinus, bilateral.* Death occurred 22 days after tooth extraction.

*Clinical Data.*—On 27 December, 1942, for reasons not stated, the lower left first molar (L-14) was extracted. There were no apparent complications. On 2 January the lower left second molar (L-15) was removed. Block anesthesia was employed on both occasions. From subsequent roentgenologic evidence it was apparent that the trouble originated in the alveolus of the lower left first molar (L-14).

On 4 January, 1943, the patient was admitted to hospital complaining of pain and swelling of the lower left jaw. He had had chills and fever. His temperature was 100.8° F., pulse 94. The gums of the left mandible were swollen and tender, and from the empty tooth sockets a moderate amount of purulent material was escaping. The subjacent cervical region was much swollen and tender and the lymph nodes could be readily palpated.

On 5 January, the fever had risen to 104.2° F., and thereafter the temperature fluctuated daily between 101.5° and 105° F., with pulse corresponding. Several chills occurred. Sulfathiazole therapy was instituted on admission; subsequent blood levels of the drug averaged 9.0 mg. per cent. By 6 January the swelling of the submandibular region was more pronounced, and appeared fluctuant. The uvula and pharynx were moderately edematous. On 7 January the patient received a transfusion of 500 c.c. of citrated whole blood. Two days later there were signs of consolidation of the right lung. The patient continued to be rational.

On 12 January the upper eyelids became edematous and there was chemosis of the conjunctivae, particularly the right. A transfusion of 400 c.c. of citrated whole blood was given. On 13 January the patient became stuporous. Two days later, 600 c.c. of fluid were aspirated from the right pleural space. Another transfusion of 400 c.c. of citrated whole blood was given. Death occurred on 18 January, 22 days after the first tooth extraction.

*Laboratory Data.*—Roentgenograms of the jaw on 7 January revealed in the region of the tooth extraction some irregularity of the periosteum and decreased density of the periapical region of the mandibular first molar (L-14) (Fig. 15). Blood examinations: on 4 January, WBC 7,700; on 5 January, WBC 24,400 (P 78, L 22); on 9 January, WBC 21,700 (P 91, L 3, M 6); on 11 January, WBC 15,000 (P 88, L 10, M 2); on 13 January, WBC 44,400 (P 79, L 19, M 2). Cultures of pleural fluid on 15 January yielded beta hemolytic streptococci. Spinal fluid examination on 12 January and blood cultures on 7 and 11 January were negative. Cultures of pus from lung abscesses post mortem yielded beta hemolytic streptococci.



Fig. 15.—Roentgenogram of the jaw taken on the eleventh day after extraction of the lower left first molar (L-14), and on the fifth day after extraction of the lower left second molar (L-15). There is a rarefied area in the periapical region of both roots of the first molar. AMM Neg. 78884.

*Pathology.*—At autopsy the fluctuant swelling in the submandibular region was found to contain 20 c.c. of pus. The suppurative process had dissected downward under the sternomastoid muscle.

In the right pleural cavity there were 300 c.c. of purulent exudate pocketed in the vicinity of the hilus near the junction of the upper and middle lobes of the lung. Both lungs were riddled with thin-walled abscesses ranging from 1.0 to 4.0 cm. in diameter. Microscopically the abscesses of the right lung were well walled off whereas those in the left lung were poorly delimited and more closely related to the bronchial tree. Other thoracic and abdominal organs showed nothing of significance.

After removal of the brain no changes were found in the skull or the paranasal sinuses. The cavernous sinuses contained "minute amounts" of purulent

exudate but no thrombus; other intracranial sinuses were normal. The meninges showed nothing of note. A large vein extending over the convex surface of the left temporal lobe contained a firmly adherent clot 1.0 cm. in length. Section of the brain revealed small abscesses in all lobes of both hemispheres. The ventricles were free from change.

*Comment.*—Because of the repeated chills and the rapid fluctuations in temperature it seems likely that pulmonary abscesses became established relatively early in the course of the illness. The abscesses doubtless were hematogenous. On the thirteenth day after extraction the right lung showed evidence of consolidation.

The bilateral conjunctival chemosis present on the sixteenth day after extraction was the first evidence of thrombophlebitis of the cavernous sinus. Thrombophlebitis of the pterygoid venous plexus as the source of the cavernous sinus thrombosis is suggested by the presence of edema of the pharynx and uvula. This is an instance, then, of cavernous thrombophlebitis in which purulent exudate at the base of the skull was absent. The brain abscesses were obviously hematogenous; the responsible organisms may have reached the general circulation either from the pulmonary abscesses or from the cavernous sinus.

CASE 13 (AMM Accession 69887).—Male, white, age 62. *Periostitis of the mandible, right; abscesses, submental and submaxillary, right; abscesses of the lungs and right kidney; brain abscesses (frontoparietal and occipital), bilateral; acute leptomeningitis.* Death occurred 64 days after extraction.

*Clinical Data.*—On 2 March, 1940, the lower right central incisor (R-9) was removed. The reason for the extraction and the mode of anesthesia are not known. The patient had had attacks of coronary thrombosis in 1935 and in 1939, and for many years had been an alcoholic. Shortly after extraction, the lower jaw became swollen and painful. Hot fomentations were applied locally in an effort to afford relief.

The patient was admitted to hospital 9 March. The pulse rate was 96. The anterior mandibular region was swollen and tender. There was severe pyorrhea, pus exuding from the necks of all the remaining lower teeth. Sulfanilamide therapy was instituted on admission; the total dosage administered is not known. On 11 March an incision was made from angle of right mandible to the submental region of the opposite side. Periostitis of the mandible and a purulent cellulitis were noted. Because of increasing peridental suppuration the following teeth were extracted: the lower incisors, cuspid and first bicuspid on the left (L-9, 10, 11, 12) and the lower lateral incisor, cuspid, and bicuspids on the right (R-10, 11, 12, 13). The operative wounds continued to drain for three weeks. The temperature ranged between 98 and 101° F.; the pulse continued to be elevated, averaging about 110.

On 4 April an intraoral incision made near the angle of the right mandible released pus. One week later some bony sequestra were removed from the right mandible by the oral route. On 13 April the submental region was incised and several more sequestra removed.

On 30 April the patient had a convulsive seizure during which his eyes deviated to the left. On 3 May a hemiplegia of the right side became evident. Death occurred 5 May, 64 days after tooth extraction.

*Laboratory Data.*—Blood examinations: on 10 March, WBC 17,000 (P 88, L 10, M 2); on 13 March, WBC 15,900 (P 71, L 19, M 9, E 1); on 24 April, WBC 15,600 (P 84, L 13, M 3); on 1 May, WBC 22,250 (P 88, L 8, M 4).



Culture of pus from the right mandibular region on 11 March yielded *Streptococcus viridans*. Blood culture on 10 April was negative.

**Pathology.**—Examination of the heart revealed an aneurysm of the left ventricle containing a laminated thrombus which had undergone pyoid softening. The aneurysm probably originated at the site of a healed infarct. The lungs contained a few small abscesses, some of which were closely associated with the bronchiolar tree. The kidneys were the seat of septic infarctions, and in the upper pole of the right kidney there was an abscess 3.5 cm. in diameter.

The leptomeninges at the base of the brain were distended with purulent exudate. Section of the brain revealed large abscesses of the frontoparietal and occipital lobes bilaterally. Smaller abscesses were present in the basal ganglia.

**Comment.**—Despite the patient's age, his previous state of ill-health, the advanced periodontal infection, the increasing osteomyelitis of the jaw, and the operative procedures, the course of the illness was slow and the bacteremia relatively late. It is of interest that although the heart was previously damaged, it was spared by the blood-borne infection.

#### BRAIN ABSCESS RESULTING FROM DIRECT SPREAD THROUGH THE CRANIUM

Brain abscess not preceded by leptomeningitis or by pachymeningitis of appreciable degree was present in three cases of the series. In all of them the infective process penetrated the greater wing of the sphenoid, or its orifices, and extended directly into the lower aspect of the temporal lobe, where abscess was produced. Thus, as regards pathogenesis they were not different from some of the others described in foregoing pages.

**CASE 14 (AMM Accession 72687).**—Male, colored, age 21. *Cellulitis of the maxillary and mandibular regions, left; abscesses, submaxillary, subtemporal, pterygopalatine, and retrobulbar, left; pericavernous suppuration with adjacent pachymeningitis, left, with development of abscess of the temporal lobe and extension to the frontal lobe, left; acute leptomeningitis; acute bronchopneumonia.* Death occurred 28 days after tooth extraction.

**Clinical Data.**—On 19 December, 1940, because of periapical abscess, the upper left first molar (L-6) was extracted. The type of anesthesia employed is not known. Several teeth were carious, and there was advanced oral sepsis.

On 27 December the patient was admitted to hospital. The temperature was 100° F. The entire left side of the face was swollen and there was sufficient pain to require morphia. Sulfathiazole therapy was instituted, 20 grains of the drug being given by mouth every four hours. On 2 January an incision was made in the left cheek, one inch behind the angle of the mouth, and a large quantity of pus was evacuated. The temperature gradually rose.

When admitted to another hospital on 11 January, the patient was semi-comatose. The temperature was 104° F., the pulse 132. Anterior and posterior cervical lymph nodes were moderately enlarged. There was complete hemiplegia of the right side, and the neck was rigid. At an unstated time a proptosis of the left eye developed. Death occurred on 16 January, 26 days after tooth extraction.

**Laboratory Data.**—Blood examination on 11 January, WBC 29,900 (P 94, L 6). Spinal fluid examination on 11 January: WBC 2,100 (P 90, L 10); culture, negative. Culture of maxillary abscess 11 January: many colonies of nonhemolytic streptococci and a few of hemolytic *Staphylococcus aureus*. Post-mortem culture of brain abscess, no growth.

**Pathology.**—Exploration of the orbit after removal of the brain disclosed a retrobulbar abscess on the left side. Orbital blood vessels and intracranial



venous sinuses were normal. A purulent exudate covered the left lateral side of the cavernous sinus and the adjoining portion of the pachymeninx. Abscesses were found in the pterygopalatine and subtemporal fossae. No information on the paranasal sinuses is available.

Examination of the surface of the brain disclosed a basilar leptomeningitis; section revealed an abscess, 2.5 cm. in diameter, in the left temporal lobe, and another, of similar size, in the adjacent part of the frontal lobe. Neither abscess had developed a capsule.

Examination of thoracic and abdominal contents revealed no abnormality other than early bronchopneumonia.

*Comment.*—The infective process, on reaching the base of the skull, spread in two directions: (1) into the retrobulbar tissues, and (2) into the lateral wall of the cavernous sinus, penetrating the meninges to invade the temporal lobe, from which the ensuing abscess extended to the frontal lobe. The proptosis was due to accumulation of pus in the orbit.

CASE 15 (AMM Accession 66700).—Male, white, age 43. *Cellulitis of the mandibular region, with development of osteomyelitis of the mandible, left; abscesses, inferior alveolar, maxillary and retrobulbar, left; osteomyelitis of the greater wing of the sphenoid, left, with overlying pachymeningitis; brain abscess (temporal lobe), left.* Death occurred 28 days after tooth extraction.

*Clinical Data.*—On 1 November, 1939, the lower four incisors (R-9, 10; L-9, 10) and the lower left first molar (L-14) were extracted. The type of anesthesia employed is not known. The teeth were removed because of advanced periodontoclasia. The gums healed well after the extraction.

About 10 November the left lower jaw became swollen and tender. The patient was admitted to hospital 13 November with temperature of 100° F. A small fistulous opening into the left jaw at the level of the lower first molar (L-14) led down to the bone. Becoming fluctuant, the swelling of the jaw was incised on 20 November, and considerable pus evacuated. Shortly thereafter a proptosis of the left eye developed. The patient's condition became progressively worse, death occurring on 29 November, 28 days after the extraction.

*Laboratory Data.*—Blood cultures on two occasions, and of the spinal fluid at autopsy, were negative. Post-mortem cultures of pus from the orbital abscess and brain abscess revealed staphylococcus, type unspecified.

*Pathology.*—On examination of the floor of the skull after removal of the brain, marked erosion of the greater wing of the sphenoid on the left side was noted. A probe inserted into this spongy area was passed easily (1) through the orbital surface of the greater wing of the sphenoid into the left orbit, where pus was encountered, and (2) through the cerebral surface of the greater wing, near the foramen ovale, into a large abscess situated lateral to the ramus of the left mandible. The dura overlying the eroded part of the sphenoid was thickened. The venous sinuses were empty.

Section of the brain revealed a small abscess at the base of the left temporal lobe adjacent to the site of bony rarefaction. Leptomeningitis was limited to the region in the vicinity of the brain abscess.

*Comment.*—After extraction, the ensuing local suppurative cellulitis extended upward to the base of the skull, visibly penetrating the latter and leading to temporal lobe abscess. En route the infection spread to the left orbit.

CASE 16 (AMM Accession 87997). Male, white, age 29. *Cellulitis and abscess of the mandibular region, right; subacute inflammation of the hypophysial capsule and the adjacent right pachymeninx; brain abscess (temporal lobe), with rupture into the lateral ventricle, right; acute leptomeningitis.* Death occurred 15 days after tooth extraction.

*Clinical Data.*—On 10 November, 1942, because of caries and periapical abscesses, the lower right first and second bicuspids and the first, second, and third molars (R-12, 13, 14, 15, 16) were extracted. The operation was performed under mandibular block anesthesia. Dental hygiene was poor.

On 12 November the patient was admitted to hospital with painful and swollen gums. The temperature was 103° F., the pulse 118. The right side of the face, especially the region at the angle of the jaw, was swollen and tender. Purulent material drained from the empty tooth sockets. By 18 November, sulfathiazole treatment was instituted; subsequent blood levels of the drug averaged 4.5 mg. per cent.

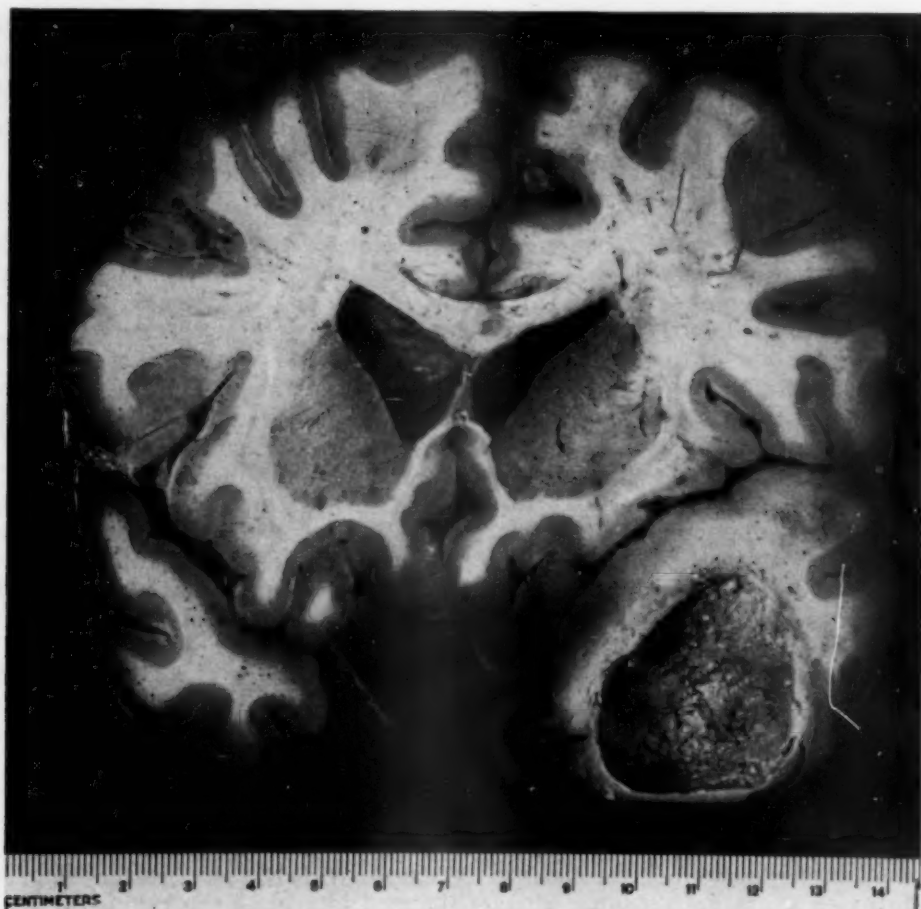


Fig. 16.—Abscess of right temporal lobe following spread of infection through the base of the skull. The abscess ruptured into the inferior horn of the lateral ventricle. AMM Neg. 76729.

On 20 November, the patient complained of headache and nausea. The temperature was 100.2° F., the pulse 84. Thereafter the pulse remained somewhat elevated but the temperature returned to normal. The swelling in the right mandibular region had acquired a fluctuant appearance. The patient seemed to be improving when, on 25 November, severe headache and vomiting set in. The left pupil was found to be larger than the right. He became drowsy, went into coma, and died. Fifteen days elapsed between tooth extraction and death.

*Laboratory Data.*—Blood examination: on 15 November, WBC 19,300 (P 85, L 12, M 3); on 23 November, WBC 12,100 (P 72, L 26, M 2). Roentgenograms of the jaw on 23 November disclosed considerable loss of bone definition accompanied by bone destruction in the area previously occupied by the first

bicuspid and the first and second molars. Smears of pus from the brain abscess post mortem revealed short-chain streptococci.

*Pathology.*—On removal of the brain, the venous and paranasal sinuses were found to be normal. The pachymeninx adjacent to the right foramen ovale had a roughened appearance. It was not studied microscopically but in the adjacent capsule of the hypophysis an infiltrate of lymphocytes intermixed with a few polymorphonuclear leucocytes was found. In the leptomeninges of the region of the left lateral fissure there was a thick yellowish purulent exudate.

The brain was edematous. On section, a moderate amount of foul-smelling purulent material was found in the right lateral ventricle. Occupying the right temporal lobe was a large cavity, which in the coronal plane measured 3.5 cm. (Fig. 16). Flaky purulent exudate adhered to its wall. At one point the wall of the abscess was contiguous with the lateral ventricle. Microscopic examination revealed early encapsulation of the abscess.

The thoracic and abdominal contents were normal except for early bronchopneumonia.

*Comment.*—Although evidence of osteomyelitis of the base of the skull is lacking, the presence of inflammatory cells in the capsule of the pituitary and the thickening of the pachymeninx of the middle cranial fossa strongly suggest that this part of the skull was traversed by the infective organism in reaching the temporal lobe.

#### CAVERNOUS SINUS THROMBOSIS

Dural sinus thrombosis was present in twelve cases in this series. One of them associated with brain abscess (Case 12) has already been described. The other eleven may be subdivided into two groups: (1) thrombosis of the cavernous and adjacent sinuses (Cases 17 to 24 inclusive), and (2) thrombosis of the lateral sinus (Cases 25, 26, and 27).

The eight cases of cavernous sinus thrombosis will be considered first. Molar teeth were extracted in all. In most of them there was suppurative cellulitis which had extended to the base or to the temporal aspect of the skull. In two (Cases 20 and 21) the cavernous sinus thrombosis was preceded by orbital abscess.

*CASE 17 (AMM Accession 62211).*—Male, white, age 44. *Cellulitis of the submandibular region, left; probable osteomyelitis of the mandible, left; abscesses, submental, sublingual, preauricular and subtemporal, left; purulent sinus thrombosis (cavernous), left.* Death occurred 20 days after tooth extraction.

*Clinical Data.*—On 14 January, 1939, the lower left second bicuspid and the adjoining first and second molars (L-13, 14, 15) were extracted. The reason for extraction and the type of anesthesia used are not known.

On the day after extraction, the patient felt feverish and had severe pain and swelling of left mandibular region. On 19 January, when he was admitted to hospital, the temperature was 100.6° F., the pulse 78. There was pronounced swelling about the left lower jaw and in the submaxillary and submental regions, but no fluctuation. Hot saline dressings were applied continuously to the affected parts.

On 21 January an incision was made intraorally, buccal to the alveolus of the lower left second molar; on the next day considerable foul-smelling pus escaped. The temperature rose to 101.6° F. On 30 January the fluctuant left submental region was incised and 5.0 c.c. of purulent material evacuated.

On 31 January there was hemorrhage (approximately 120 c.c.) into the mouth originating from the alveolus of the lower left second molar, following



which the patient had a chill lasting twenty-five minutes. Cellulitis made its appearance over the entire left side of the face, the postauricular region, and the adjoining scalp. Incision of the left temporal fossa released considerable pus. During that day the left eyelid became increasingly edematous, and the eyeball more and more prominent; there was slight papilledema but the fundal veins were said not to be engorged. The pupils remained equal and reactive. Sulfanilamide therapy in doses of 9 Gm. daily, was started. On 2 February stupor set in. A small fluctuant area above the left ear was incised. Death occurred on 3 February, 20 days after the extraction.

**Laboratory Data.**—Blood examination on 21 January, WBC 21,850 (P 89, L 11), and on 23 January, WBC 15,200 (P 80, L 19, M 1). Spinal fluid examination on 2 February, WBC 150 (P 85, L 15). Culture of pus from the submental abscess on 30 and 31 January disclosed gram-positive diplococci, gram-negative bacilli and a few colonies of *Staphylococcus aureus*; blood culture on 31 January yielded the same types of diplococci and bacilli and, in addition, anaerobic streptococci. Spinal fluid culture on 2 February was negative.

**Pathology.**—The only intracranial lesion of note consisted of a frankly purulent thrombus in the left cavernous sinus. The other venous sinuses were empty. The leptomeninges and brain were normal, as were also the paranasal sinuses. The thoracic and abdominal viscera were free from significant change.

**Comment.**—The cavernous sinus thrombosis occurred on the seventeenth day after extraction. Gaining the cavernous sinus, the organism was swept into the general circulation but no visceral lesions resulted. The spread to the sinus was doubtless through venous channels penetrating the skull.

**CASE 18 (AMM Accession 41097).**—Male, Filipino, age 39. *Osteomyelitis of the mandible with cellulitis, first left and then right, with spread to the right temporal region; pulmonary abscesses, bilateral; thrombosis of the ophthalmic vein, left; sinus thrombosis (cavernous, circular), bilateral; acute leptomeningitis.* Death occurred 21 days after tooth extraction.

**Clinical Data.**—On 18 September, 1932, the patient entered hospital because of pain and swelling of the left side of the face. A periodontal abscess was suspected. There was severe pyorrhea. Roentgenograms showed rarefaction and multiple sequestra of the left mandible. On 19 September the lower left first, second, and third molars (L-14, 15, 16) were extracted. The type of anesthesia employed is not known. Soon a cellulitis of the mandibular region became evident, first on the left and then on the right. The cervical lymph nodes became moderately enlarged, and remained so. On 23 September and again on 27 September, incisions were made over both mandibles, and considerable pus encountered.

On 2 October a fluctuant area in the right temporal region was incised and drained. Four days later a proptosis of the left eye developed. Previously of low grade, the fever rose to 104° F., and there were repeated chills. On 9 October the neck became stiff. Death occurred on 10 October, 21 days after tooth extraction.

**Laboratory Data.**—Blood examination on 23 September: WBC 19,000 (P 92, L 8). Spinal fluid (date not given): WBC 87 (P 98, L 2).

**Pathology.**—Examination of the thoracic contents revealed abscesses in the lower lobes of both lungs. The abdominal organs were normal. Tissues of the face, especially the left masseter and left temporal muscle, were necrotic. Osteomyelitis of the mandible was evident bilaterally.

Exploration of the venous sinuses revealed purulent thrombosis of both cavernous sinuses and of the circular sinus. The left ophthalmic vein also contained a septic thrombus. The leptomeninges, particularly those of the base of the brain, were the seat of purulent exudate. There was no gross involvement of the brain.



*Comment.*—Tooth extraction was performed in an attempt to provide drainage of the osteomyelitic mandible but it served only to intensify the condition. At an early stage the infective organism entered the blood stream, leading to the formation of pulmonary abscesses. Thrombosis of the cavernous sinus occurred on the eighteenth day after extraction, and leptomeningitis on the twentieth. Presumably both were due to direct spread of the infection through the base of the skull.

CASE 19 (AMM Accession 103922).—Male, white, age 31. *Gingivitis, right mandible; cellulitis of the pharynx and cervical region, first right and then left, and of the temporal region, right; bacteremia, with development of pulmonary abscesses, right; sinus thrombosis (cavernous), bilateral; purulent sphenoidal sinusitis; acute leptomeningitis; moderate subarachnoid hemorrhage, base of brain.* Death occurred 23 days after tooth extraction.

*Clinical Data.*—The lower right first molar (R-14) was extracted 14 November, 1943, under local infiltration anesthesia because of severe toothache which had started the day before. A few hours later the patient complained of headache and throbbing pain in the region of the extraction; fever and chills followed.

The patient was prostrated when admitted to hospital on 19 November, the temperature was 103° F., the pulse 112. A scanty discharge issued from the gaping tooth socket. The surrounding gum was red and edematous. The right side of the face as high as the zygomatic arch was indurated and sensitive to pressure; the swelling had spread also to the upper right cervical region. The submaxillary lymph nodes on the right side were somewhat enlarged. By 20 November, the cellulitis had spread to the pharynx and to the upper cervical region of the left side. On the next day, proptosis and conjunctival edema of the right eye became apparent, and soon the left eye became similarly affected. On 25 November there were palsies of the right oculomotor and abducent nerves as well as hypesthesia in the region of distribution of the ophthalmic division of the trigeminal nerve.

Gradually the bilateral chemosis, the proptosis, and the swelling of the neck receded; however, the right extraocular palsy became more severe, now including the trochlear nerve. The right pupil became permanently dilated. Despite repeated whole blood and plasma transfusions and sulfadiazine (with average of 11.0 mg. per cent in blood stream) and penicillin therapy (total dose 1,700,000 units) which had been given since 23 November, the patient failed to improve. On 4 December the neck became rigid. Death occurred on 7 December, 23 days after tooth extraction.

*Laboratory Data.*—Blood leucocytes ranged from 14,900 (P 81, L 19) on 19 November to 46,000 (P 88, L 12) on 25 November and 22,300 (P 89, L 11) on 7 December. Spinal fluid on 4 December was xanthochromic and contained 650 leucocytes (P 98, L 2); culture was negative. Blood cultures on 19 November and at autopsy were positive for nonhemolytic streptococci.

*Pathology.*—In all lobes of the right lung there were a number of small abscesses, most of them related to the bronchiolar tree. The largest (1.0 cm. in diameter) was well encapsulated.

A leptomeningeal hemorrhage of undetermined origin was found at the base of the brain and on the ventral surface of the pons. It was most abundant in the submamillary region (Fig. 17). In addition there was acute leptomeningitis. The posterior cerebral artery contained an early mural thrombus and the walls of pontine veins were invaded by inflammatory cells. The brain itself showed nothing of note.

Examination of the venous sinuses revealed a thrombosis of both cavernous sinuses, the right being the more extensively involved. The sella turcica, especially its posterior portion, was eroded, and the subjacent sphenoidal sinus contained blood-tinged mucopurulent material. The mucosa of the ethmoidal sinus was thickened. The other paranasal sinuses were normal.

*Comment.*—The histologic appearance of the right lung suggests abscess formation in the early course of the illness with subsequent dissemination by way of the bronchial tree. Bacteremia was demonstrated on the fifth day after extraction. The intracranial involvement does not, however, appear to have been on a hematogenous basis. The cavernous sinus thrombosis, which occurred

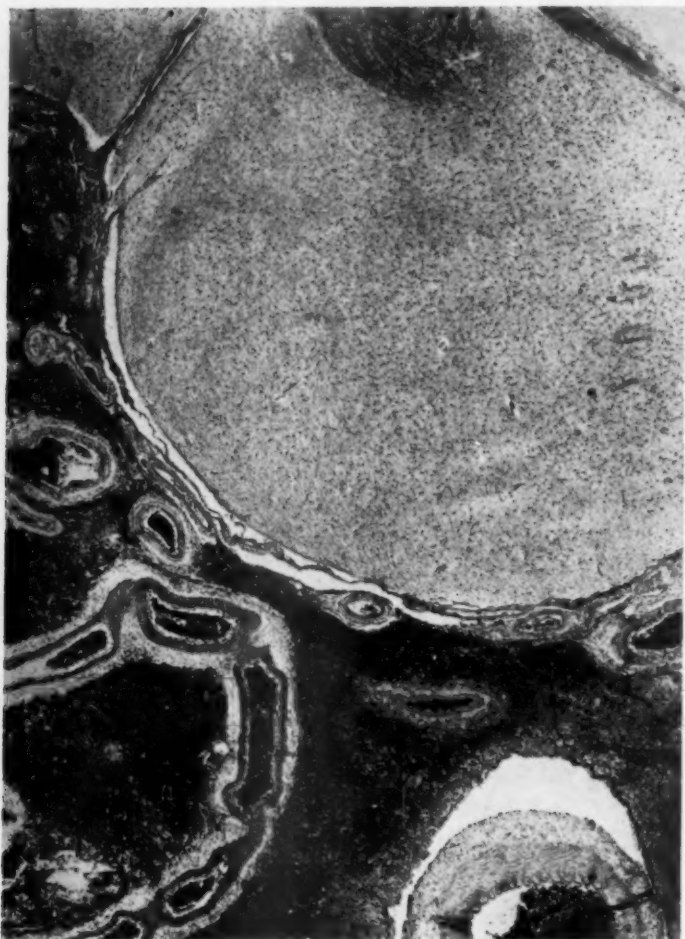


Fig. 17.—Subarachnoid hemorrhage at the base of the brain in the region of the mamillary body. The mamillary body is clearly visible. There is also purulent leptomeningitis of moderate degree. AMM Neg. 78747. Hematoxylin and eosin stain.  $\times 20$ .

on the eighth day after extraction, may have been a consequence of spread of the infection to the base of skull, involving the sphenoidal sinus and presumably also the pterygoid venous plexus.

**CASE 20** (AMM Accession 39193).—Male, white, age 34. *Cellulitis of the left maxillary region; abscesses, subtemporal and retrobulbar, left; purulent sinus thrombosis (cavernous), left, with adjacent dural abscess; subarachnoid hemorrhage, left frontal lobe.* Death occurred 12 days after tooth extraction.

**Clinical Data.**—On 1 December, 1932, for reasons not known, the upper left third molar (L-8) was extracted. The type of anesthesia employed is not recorded. Shortly after the extraction the left maxillary region began to swell.

On 3 December the patient was admitted to hospital with temperature of 99.8° F., and pulse of 80. The submaxillary lymph nodes of the left side were somewhat enlarged, and the tissues of the left maxillary region indurated.

Hot compresses were applied locally. On 5 December the left eye became prominent, and on the next day it bulged considerably. The temperature, previously within normal limits (except on admission), had risen to 100.6° F.; thereafter it ranged between 99 and 100° F. On 9 November an abscess in the region of the lower left third molar (L-16) was incised and drained intraorally.

On 10 December, because of fluctuation in the region of the inner canthus of the left eye, incision was made into the left orbit and about 4.0 c.c. of pus evacuated. On the next day there was hemorrhage at the site of the intraoral incision, approximately 150 c.c. of bloody pus escaping. Shortly thereafter the patient became stuporous. Death occurred 12 December, 12 days after tooth extraction.

*Laboratory Data.*—Blood examination on 7 December, WBC 21,200 (P 88, L 12); and on 10 December, WBC 13,300 (P 87, L 13). Roentgenograms on 8 December showed increased density of the left maxilla and zygoma; in addition there was a shadow consistent with intraorbital abscess. Post-mortem cultures of pus from the left maxillary region yielded staphylococci, and from the left cavernous sinus gram-negative diplococci which were identified as meningococci and regarded as a secondary invader.

*Pathology.*—On removal of the brain the left cavernous sinus was found to contain a purulent thrombus. The adjacent dural surface was covered with a thick layer of pus. Removal of the greater wing of the left sphenoid and the orbital plate of the frontal bone revealed pus around the layer of fatty tissue behind the eye. The brain showed nothing of significance. The leptomeninges of the convex surface of the frontal lobe were bloody.

*Comment.*—The infective agent spread rather rapidly from the maxilla to the subtemporal fossa, thence gained the back of the orbit, producing an abscess. The orbital abscess doubtless was the source of the purulent cavernous sinus thrombosis.

CASE 21 (AMM Accession 74324).—Male, colored, age 44. *Gingivitis with abscess of the alveolus of the lower right third molar; abscesses of both orbits, first right and then left; sinus thrombosis (cavernous, circular, inferior petrosal), bilateral; purulent sphenoidal sinusitis; pulmonary abscesses, bilateral; otitis media, left; brain abscesses (temporal and parietal), left.* Death occurred 36 days after tooth extraction.

*Clinical Data.*—The patient came to the clinic for treatment of swollen and painful gums, and on 25 February, 1941, for reasons not stated, the lower right third molar (R-16) was extracted. The type of anesthesia employed is not known. On the next day the gum in the region of extraction became swollen. On 4 March the fluctuant alveolus R-16 was incised, and foul-smelling pus drained.

On 12 March the right eye, and then the left, began to bulge. The patient's general condition grew progressively worse, and on 17 March he was admitted to hospital in semicoma. There was severe anemia. The temperature was 97° F., the pulse 92. The cervical lymph nodes of both sides were moderately enlarged. The inner canthus of the left upper eyelid was oozing pus, while that on the right was fluctuant and about to rupture. Under ether anesthesia, both orbits were incised and drained. On 21 March sulfathiazole therapy was instituted, the subsequent blood levels of the drug averaging 4.0 mg. per cent. Two blood transfusions were given. On 24 March the temperature reached 100° F., the pulse 116.

By 25 March there was bilateral ophthalmoplegia, flaccid hemiplegia of the entire right side, and moderate stiffness of the neck. The right post-mandibular abscess continued to drain freely into the mouth. On 28 March the left orbit was again incised and drained. Death occurred on 2 April, 36 days after extraction.



**Laboratory Data.**—Blood counts: on 12 March, RBC 2,660,000, WBC 16,550 (P 82, L 18); on 17 March, RBC 3,150,000, WBC 11,000 (P 85, L 8, M 7); on 20 March, RBC 2,430,000, WBC 12,250 (P 72, L 24, M 3, B 1); on 1 April RBC 2,840,000, WBC 13,550 (P 86, L 12, M 2). Blood culture on 27 March: no growth. Culture of pus from left orbit on 28 March: heavy growth of gram-negative diplococci, type unidentified. Spinal fluid culture on 29 March: no growth.

**Pathology.**—Examination of the thoracic contents revealed a well-encapsulated abscess of the lower lobe of the right lung, 4.0 cm. in diameter, and numerous early abscesses throughout both lungs, most of them about 0.5 cm. in size. The other viscera were free from change.

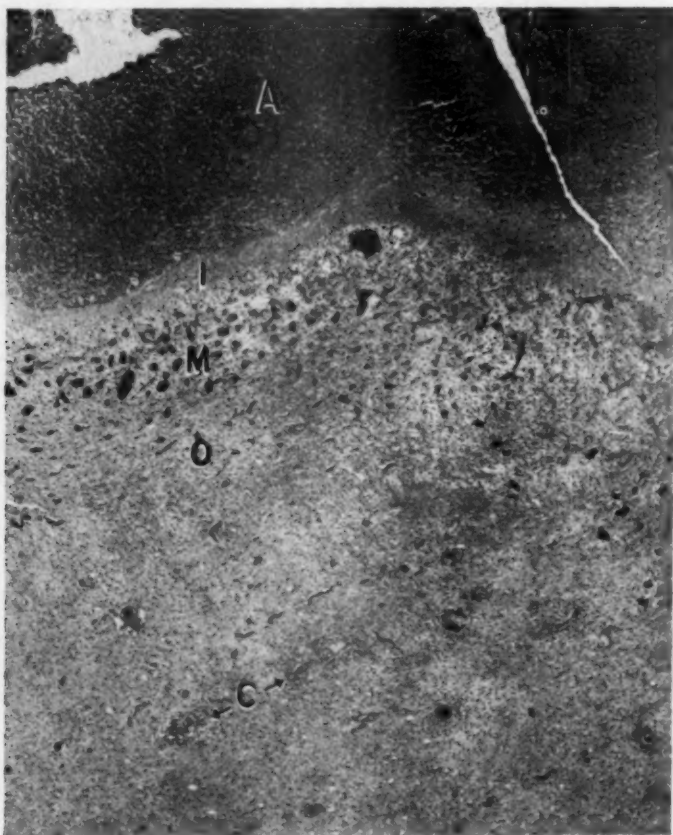


Fig. 18.—The wall of an abscess of the left parietal lobe. The wall of the abscess (A) is composed of three layers: an inner (I), consisting of collagenous connective tissue fibers, a middle (M), made up of well-vascularized fibroblastic tissue in which many histiocytes, lymphocytes and plasma cells are present, and an outer (O), composed of connective tissue containing histiocytes, fibroblasts, and other cells. Perivascular cuffs of lymphocytes (C) are present in the surrounding brain. AMM Neg. 83739. Hematoxylin and eosin stain.  $\times 40$ .

Purulent thrombi were found in the cavernous, the circular, and the inferior petrosal sinuses bilaterally. The subdural space was dry. Both orbits contained abundant pus, as did also the right sphenoidal sinus. The ethmoidal sinuses had a dull but nonpurulent lining. Pus was found in the left middle ear and in the immediately adjacent bone.

The leptomeninges showed nothing of note. Section of the brain revealed four deep-seated abscesses in the left parietal and temporal lobes, three of which had coalesced. The largest abscess,  $5 \times 4 \times 1.5$  cm. in size, had a capsule of moderate thickness (Fig. 18).



*Comment.*—The abscess of the right orbit occurring on the fifteenth day after extraction may be assumed to have been due to upward spread of the infection through the inferior orbital fissure. The pathogenesis of the left orbital abscess is not evident: perhaps the source of the abscess was the purulent sphenoidal sinus.

The sphenoidal sinusitis was an unanticipated finding. Its origin is uncertain. There was no osteomyelitis of the adjacent greater wing of the sphenoid. One takes into consideration the possibility that the cavernous sinus thrombosis was due to spread of an infective process from the middle ear to the jugular bulb, then via the inferior petrosal sinus to the cavernous (as in cases reported by Turner and Reynolds, and Eagleton) but it seems more logical to assume that the cavernous sinus thrombosis was a complication of orbital abscess. The sphenoidal sinusitis may represent a forward spread through veins which drain the mucosa of the sphenoidal sinus into the cavernous sinus (a route of spread substantiated by Turner and Reynolds).

For want of evidence of direct spread, the otitis media and the brain abscesses may be regarded as hematogenous.

CASE 22 (AMM Accession 93087).—Male, white, age 28. *Cellulitis of the right cheek; osteomyelitis of maxilla, nasal bone, greater wing of sphenoid and squamous part of the temporal bone, first on the right side and then on the left; suppurative pansinusitis; abscesses, maxillary, periorbital and temporal, left, and epidural and perihypophysial, bilateral; thrombosis of the angular vein, right; sinus thrombosis (cavernous, circular, petrosals, lateral), bilateral; early subdural empyema; acute leptomeningitis.* Death occurred 48 days after extraction.

*Clinical Data.*—Because of recurrent headaches and nasal obstruction, polyps were removed from the left nostril in August and in early November 1942, and from both antra on 12 November. Recovery was uneventful. Then on 20 November, because of roentgenologic evidence of periapical abscess, the upper right first molar (R-6) was extracted. The type of anesthesia employed is not known.

On admission to hospital 22 November there was exquisite tenderness of the right cheek. Other than the swelling at the site of extraction nothing of significance was found. The temperature was 99° F. Sulfathiazole therapy was instituted, and continued throughout the subsequent course of the illness. The blood concentration of the drug averaged 6.0 mg. per cent.

On 23 November the temperature reached 101.4° F., the pulse 96. Irrigation of the right maxillary sinus failed to reveal pus. The swelling spread to the region of the right eyelids and the right side of the nose. A clinical diagnosis of thrombosis of the right angular vein was made. At this time a purulent discharge, presumably from the ethmoidal sinuses, began to drain from both nostrils. On 25 November an intraoral incision was made above the right canine fossae and a drain inserted. Five days later the right antrum was opened through the canine fossa.

Soon it became evident that the cellulitis was spreading to the left side of the face. On 2 December a small abscess in the left palatal region was incised and drained. Two days later, when the temperature was 104° F., a transfusion of 500 c.c. of citrated whole blood was given. (Subsequently the patient received five more transfusions.) On 8 December, the now fluctuant right lower eyelid was incised. On 9 December the left upper cuspid (L-3)

was extracted. Five days later, multiple incisions were made intraorally on the left side. One of these opened the left maxillary sinus. Irrigation of the sinus with sulfanilamide in saline was begun. On 15 December, because of loosening due to osteomyelitis, the upper left third molar (L-8) was extracted, and on 21 December the upper left first and second molars (L-6, 7).

On 23 December the patient was transferred to another hospital where further surgical procedures were carried out: incision and drainage of the left maxillary sinus through the canine fossa, left external ethmoidectomy, and incision and drainage of the left temporal fossa. At this time the temperature fluctuated between 100 and 103° F.

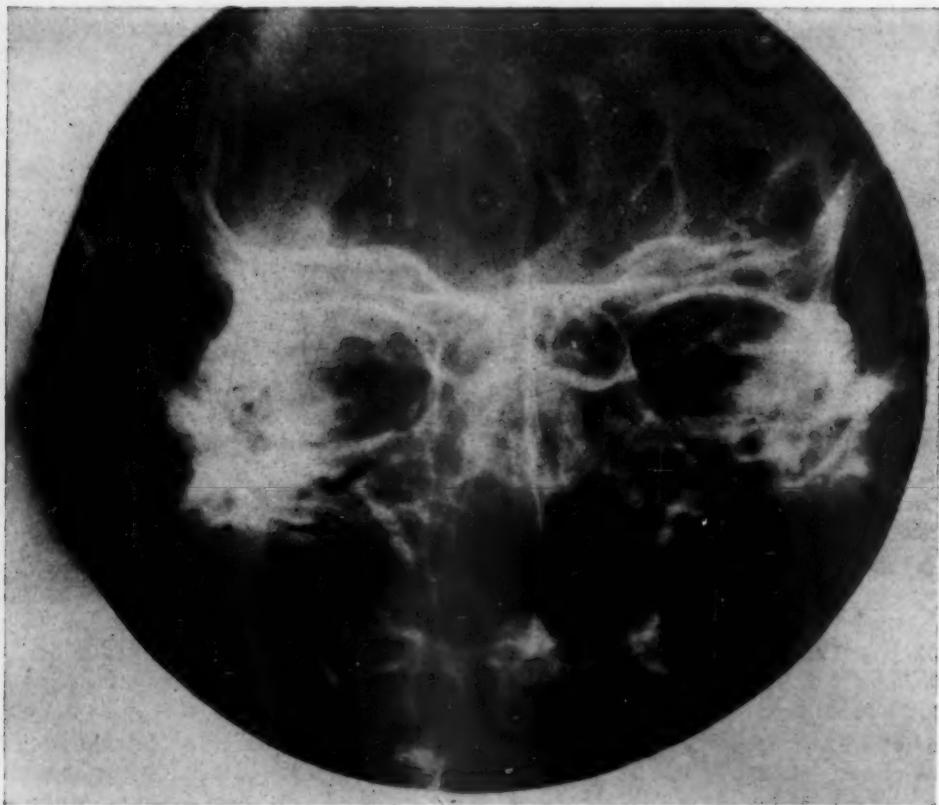


Fig. 19.—Roentgenogram of the skull on the thirty-fourth day after extraction of the upper right first molar (R-6). There is considerable clouding of the ethmoidal, sphenoidal, and right maxillary sinuses. AMM Neg. 78989.

On 25 December the conjunctivae of the right eye became edematous and soon the eyeball protruded. Heparin therapy was of no noticeable benefit. On 6 January the tissues of the neck along the course of the jugular veins became tender. Toward the end there was equivocal evidence of meningitis. Death occurred 7 January, 48 days after tooth extraction.

**Laboratory Data.**—Blood examinations; on 4 November, RBC 4,500,000, WBC 8,000 (P 72, L 20, E 8); on 27 November, WBC 13,150 (P 67, L 33); on 1 December, WBC 19,800 (P 78, L 19, E 3); on 10 December, WBC 6,600 (P 66, L 27, M 3, E 4); on 19 December, WBC 21,450 (P 72, L 25, M 3). Roentgenograms on 24 December showed cloudiness of all paranasal sinuses and evidence of osteomyelitis of facial bones (Fig. 19). Cultures of pus from the left ethmoidal sinuses (1 January) yielded hemolytic *Staphylococcus aureus*. Three blood cultures (25 and 26 December, and 6 January) showed no growth. Post-

mortem cultures of heart blood, and of pus from the right maxillary region and the base of the brain each yielded *Streptococcus viridans*.

**Pathology.**—After the brain was removed, an examination of the base of the skull revealed large epidural abscesses of the middle cranial fossae. The underlying bone, including the sella turcica, the greater wing of the sphenoid, and the squamous part of the temporal bone, had a moth-eaten appearance and exuded considerable pus. More posteriorly, the adjoining petrous part of the left temporal bone contained pus pockets but there was no visible involvement of the mastoid air cells. The intraorbital tissues were not affected. The frontal, maxillary, ethmoidal, and sphenoidal sinuses of both sides contained free pus.

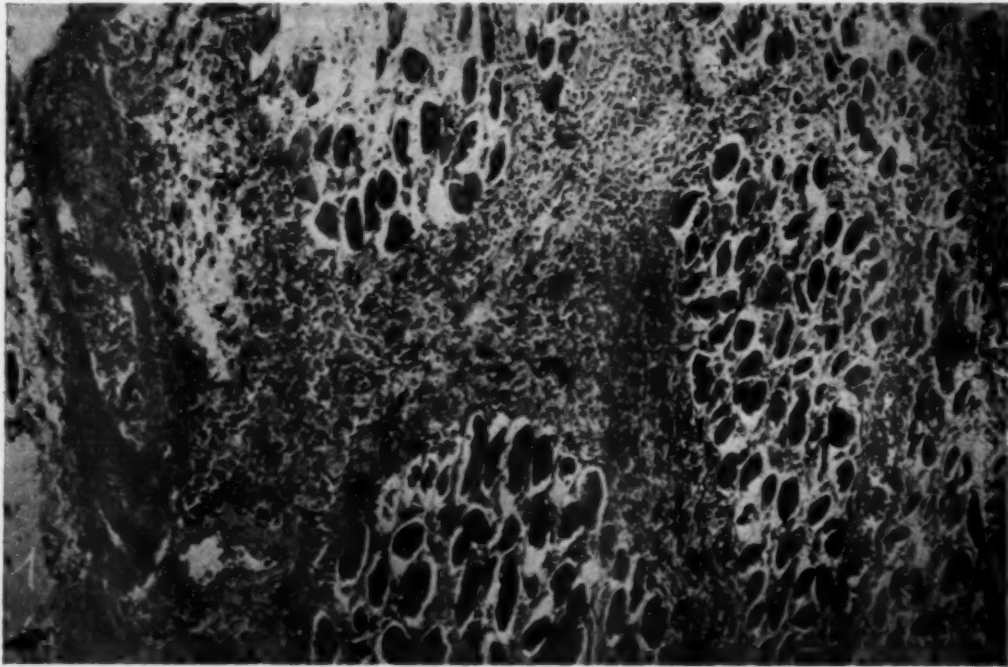


Fig. 20.—Acute cellulitis of the constrictor muscle of the pharynx. The section is from the region about 1.0 cm. beneath the pharyngeal mucosa. There is heavy infiltration of polymorphonuclear leucocytes in the connective tissue around and within muscle bundles. Some muscle fibers have undergone degeneration. AMM Neg. 84320. Hematoxylin and eosin stain.  $\times 150$ .

Investigation of the dural sinuses disclosed creamy pus in both cavernous sinuses. Less purulent thrombi were found also at the junction of superior petrosal and lateral sinuses of both sides. The internal jugular veins were not explored.

The leptomeninges covering the base of the brain and anterior aspect of the brain stem contained patchy yellowish exudate. Section of the brain showed nothing of significance.

Microscopic examination confirmed most of the gross findings; muscles were the seat of purulent cellulitis (Fig. 20); the mucosae of paranasal sinuses were edematous and congested, and some of the veins are filled with pus (Figs. 21 and 22); bones at the base of the skull were purulent, and veins of the overlying dura contained well-organized thrombi (Fig. 23); the cavernous sinuses contained purulent exudate and the superior petrosal sinuses were filled with a fibrinopurulent thrombus (Fig. 24); leptomeningeal veins on the convex surface of the brain as well as those of the ventral surface of the pons were distended with pus (Fig. 25).



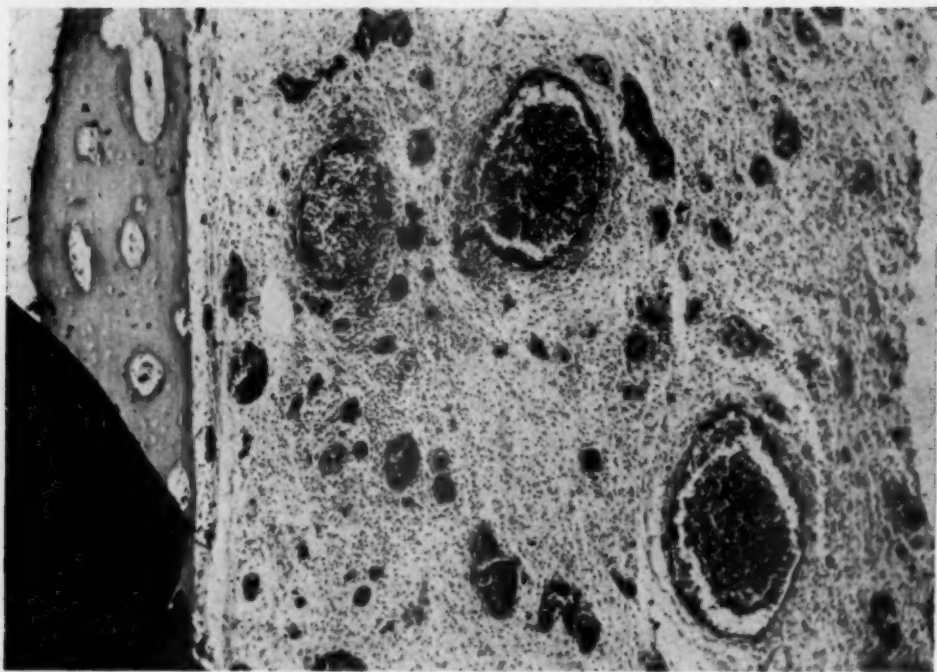


Fig. 21.—Purulent phlebitis of the mucous membrane of the sphenoidal sinus. In the mucosa, which is markedly congested, there are three veins filled with pus. AMM Neg. 84322. Hematoxylin and eosin stain.  $\times 100$ .



Fig. 22.—Purulent sinusitis and osteomyelitis, sphenoidal. The thickened mucosa (M) of the sphenoidal sinus is disrupted by purulent exudate. The adjacent bone shows osteomyelitis; the pus extends through the bone (X) and burrows into the overlying dura (D). The thrombosed vein (V) in the dura is shown at greater magnification in Fig. 23. AMM Neg. 78738. Hematoxylin and eosin stain.  $\times 25$ .



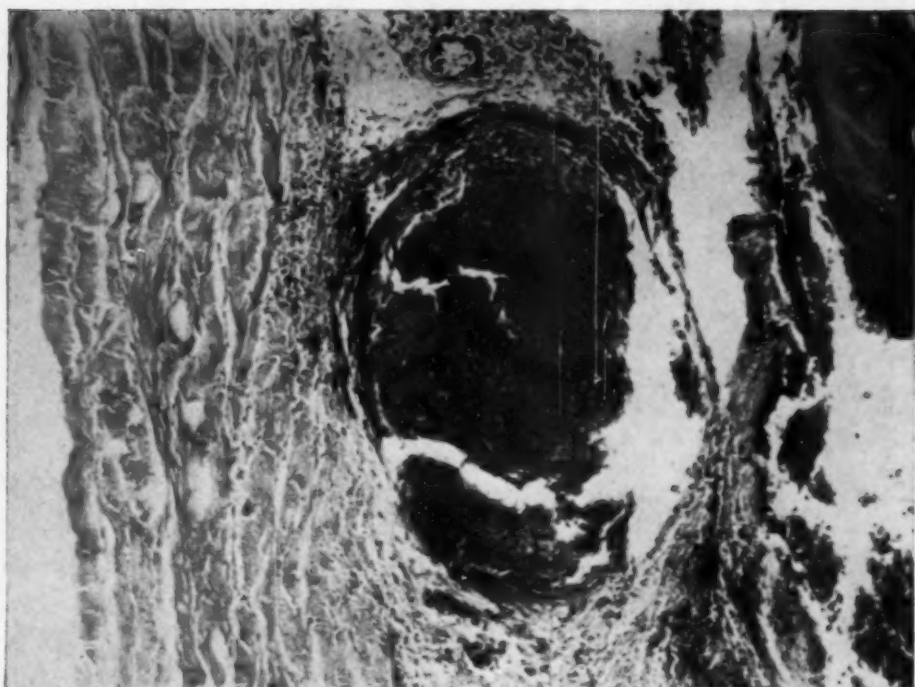


Fig. 23.—Thrombosis of vein traversing the dura of the sphenoid bone. The thrombus is well organized. The relations of the vein are shown in Fig. 22. AMM Neg. 84327. Hematoxylin and eosin stain.  $\times 175$ .

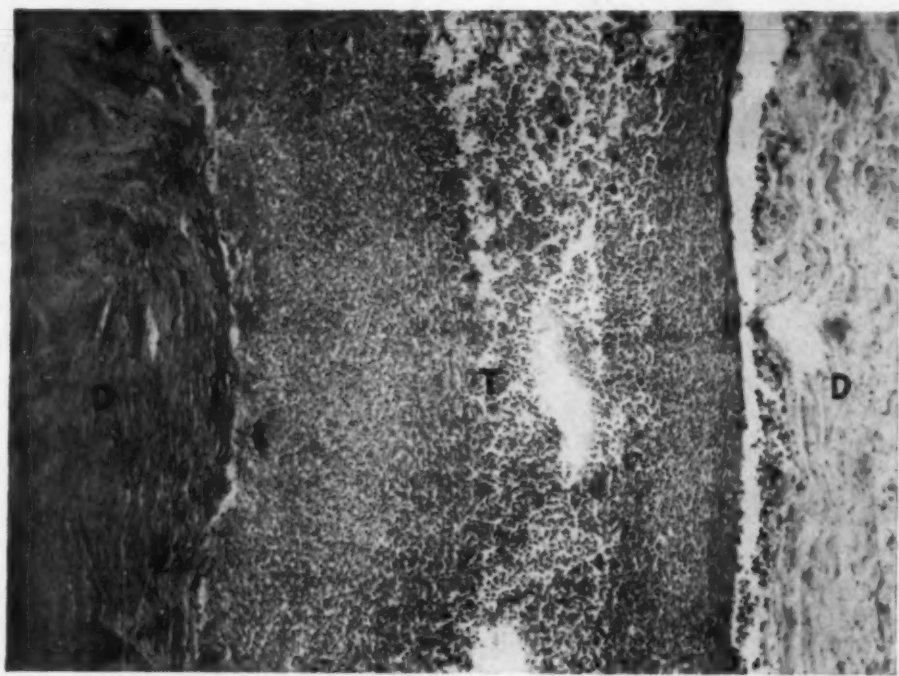


Fig. 24.—Thrombosis of superior petrosal sinus. The thrombus (T) is fibrinopurulent. The adjacent dural wall (D) is relatively normal. AMM Neg. 84323. Hematoxylin and eosin stain.  $\times 100$ .

*Comment.*—Despite all the efforts to drain purulent foci, the infective agent continued to spread until most of the base of the skull was osteomyelitic. Thirty-six days passed before the cavernous sinuses became thrombosed, and not until shortly before death, was the dural barrier penetrated. A point of passing interest is the presence of thrombosed veins in the dura adjacent to the cavum sellae. This thrombosis probably is continuous with that in the cavernous sinus.

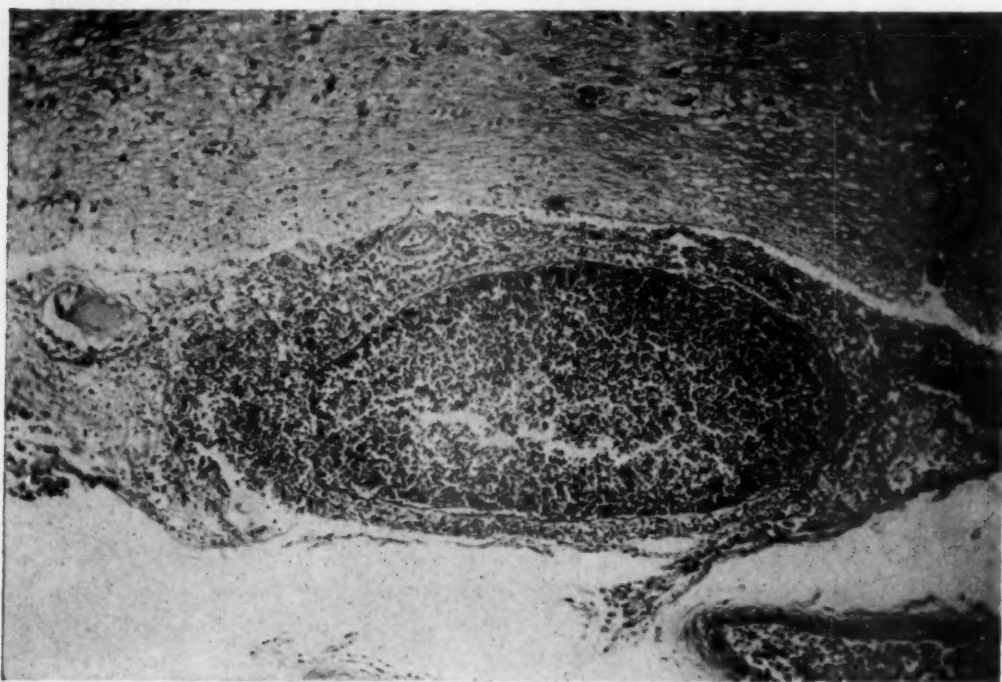


Fig. 25.—Purulent thrombophlebitis of leptomeningeal vein at the base of the pons. The lumen is distended with pus. AMM Neg. 84319. Hematoxylin and eosin stain.  $\times 100$ .

There are three instances (Cases 22, 19, and 18) in this series of twenty-eight in which suppurative cellulitis starting on one side of the jaw extended to the other side. In the present case it seems likely that the spread to the contralateral side was by way of the base of the skull since at about the time of spread an abscess formed in the palate and there was profuse purulent discharge into both nostrils, presumably from ethmoidal sinuses. Similarly in Case 19 the passage of the infection to the opposite side (and thence down into the neck) was by way of the posterior route, as judged by preliminary cellulitis of the pharynx. In Case 18 the infection crossed the midline as periostitis of the mandible, and thereupon extended through soft tissues to reach the temporal region.

In two of eight cases of cavernous sinus thrombosis described by Childs and Courville there was likewise a spread to the side opposite the dental infection. The routes pursued were not defined but the authors regarded retrograde thrombosis of the pterygoid venous plexus subsequent to cavernous sinus thrombosis as a feasible means of spread.

CASE 23 (AMM Accession 61687).—Female, white, age 42. *Cellulitis with abscess formation, posterior maxillary and upper cervical regions, right; severe*

osteomyelitis of the sella turcica and of the adjacent portion of the greater wing of the sphenoid, and of the petrous part of the temporal bone, right; acute leptomeningitis; sinus thrombosis (cavernous, circular, petrosals, lateral), bilateral. Death occurred 28 days after tooth extraction.

*Clinical Data.*—On 17 February, 1939, because of periapical abscesses, the upper right second and third molars (R-7, 8) were extracted. The type of anesthesia employed is not known. There was pyorrhea of moderate degree. After extraction, the jaw became unduly tender, and on the fourth day the face began to swell.

On admission to hospital 25 February the temperature was 99.4° F., the pulse 95. By 28 February the swelling of the right side of the face had become considerably worse. Sulfanilamide therapy was begun, approximately 4.0 Gm. of the drug being given daily. On 6 March an abscess in the upper posterior maxillary region ruptured spontaneously into the mouth. Low-grade fever persisted. On 8 March, because of cyanosis, headache, and generalized pains, the use of sulfanilamide was discontinued. Two days later the patient's condition had improved and the swelling of the face had receded, but drainage from the abscess into the mouth was still abundant. On 14 March, two loose teeth (lower right first and second molars: R-14, 15) were extracted.

On 15 March, stupor set in. There was a semipurulent nasal discharge, most profuse on the right. The tooth sockets of R-7, 8, 14, and 15 exuded pus. Swelling of the right side of the face again had become prominent. The neck was stiff and the Kernig sign positive. Sulfanilamide by mouth was resumed, and neoprontosil given intraspinally. On 16 March the conjunctivae of both eyes became congested and edematous. Death occurred on 17 March, 28 days after tooth extraction.

*Laboratory Data.*—Blood examinations: on 25 February, WBC 9,000 (P 71, L 29), and on 5 March, WBC 25,000 (P 93, L 7). Spinal fluid examination on 15 March: WBC 6,500 (P 90, L 10); colloidal gold curve 0002223333. Blood culture on 15 March, no growth. Culture of maxillary abscess on 12 March, *Streptococcus hemolyticus* and *viridans*; of maxillary abscess, blood, and urine post mortem, *Streptococcus viridans*.

*Pathology.*—Examination of the skull after removal of the brain disclosed severe osteomyelitis of the sella turcica and of the adjacent portion of the greater wing of the right sphenoid. There was also considerable pus in the petrous portion of the right temporal bone. Adherent blood clots were found in most of the dural sinuses (cavernous, circular, petrosals, lateral) of both sides. Some of the cerebellar veins contained agonal clots while others were filled with leucocytes. The paranasal sinuses were not examined. A heavy purulent exudate was present in the cerebral and spinal leptomeninges. Section of the brain revealed nothing of significance.

*Comment.*—Inasmuch as sinus thrombosis was a terminal event, it seems likely that it was secondary to invasion of the sphenoid bone by the infective organism.

CASE 24 (AMM Accession 50939).—Male, white, age 24. *Cellulitis of the left mandibular region; peritonsillar abscess, left; sinus thrombosis (cavernous, superior petrosal, lateral, sigmoid), bilateral; massive subdural hemorrhage.* Death occurred 5 days after tooth extraction.

*Clinical Data.*—On 18 August, 1936, because of pain, the erupting lower left third molar (L-16) was extracted. Conduction anesthesia with procaine was employed.

Owing to an attack of fainting, the patient was admitted to the hospital two and one-half hours after the extraction. He felt weak and nervous and had a fear of impending death. His pulse was fast and thready. Already



there was considerable induration of the face in the region of the left mandible. Trismus was marked.

From the left posterior mandibular region the swelling spread to the pharyngeal tissues and to the cervical region. On 20 August the patient was found walking aimlessly about the ward. His temperature on that day was 101.6° F., the pulse 100. On 22 August the pharynx became still more edematous. Prostration grew steadily more severe. Cyanosis became evident. The temperature rose to 102° F., the pulse to 120. An incision was made just in front of the left tonsil, and a considerable amount of pus evacuated. Death occurred on 23 August, 5 days after tooth extraction.

*Laboratory Data.*—Urinalysis on 19 August, negative. Blood count on 23 August: RBC 4,100,000; Hb. 80 per cent; WBC 24,300 (P 91, L 6, M 1, E 2).

*Pathology.*—Removal of the brain disclosed a massive subdural hemorrhage, most profuse over the base of the brain and around the brain stem, and slight hemorrhage in the leptomeninges. The hemorrhage was judged to be relatively recent. Exploration of the dural sinuses revealed bilateral thrombosis of the cavernous, the superior petrosal, the lateral, and the sigmoid. The thrombi, found to be adherent to the walls of the sinuses, were continuous down to the jugular foramina. Section of the brain revealed nothing of note.

*Comment.*—Cellulitis of the posterior part of the left mandible spread rapidly to the pharynx, and by the fifth day had developed into an abscess. The symptomatology is difficult to evaluate. It may be argued that the infective process reached the cavernous sinuses by way of postpharyngeal venous tributaries which extend upward through the base of the skull to drain into the cavernous sinus (Fig. 32), and that once established in the cavernous sinus, the thrombus increased in length, coming to occupy most of the other sinuses. Subdural hemorrhage could account for the rapidly fatal outcome. It is doubtful that bacteremia could have been responsible for this train of events.

#### LATERAL SINUS THROMBOSIS

Thrombosis restricted to the lateral (or transverse) sinus was found in three instances. In one of these (Case 25) the pathogenesis was obvious: the infective process had spread backward to the petrous part of the temporal bone, then penetrated the adjacent dural barrier. In another (Case 26), bilateral in distribution, in which the thrombosis of one side was complicated by cerebellar abscess, the mode of spread was not evident but was considered to be via the temporal bone. In the third (Case 27), also bilateral, the cause of the thrombosis is not known but is thought to be the result of blood-borne infection.

*CASE 25 (AMM Accession 96492).*—Male, white, age 35. *Cellulitis of the mandibular region, left; abscesses, submandibular and subtemporal, left; osteomyelitis, mandible, sphenoid and temporal (squamous and petrous), left; mastoiditis, with epidural abscess and sinus thrombosis (lateral), left; acute leptomeningitis, with ependymitis and choroiditis of the fourth ventricle; acute bronchopneumonia.* Death occurred 136 days after tooth extraction.

*Clinical Data.*—In April, 1942, the patient burned his left index finger, and because of a complicating "streptococcus" infection was hospitalized for two weeks. A subsequent reinfection of the finger kept him in bed for another three weeks. In November he was admitted to hospital for treatment of long-



standing recurrent prostatitis and urethral discharge. Chronic pleurisy also was in evidence. Six weeks later the conditions, regarded as nonspecific, had greatly improved.

Because of the recurring infections the patient was examined again. Numerous teeth were found to be carious, some of them impacted. On 4 January, 1943, the upper and lower right third molars (R-8, 16) were extracted. Uneventful recovery ensued. Then on 27 January the corresponding molars on the left side (L-8, 16) were removed. The type of anesthesia employed is not known. The lower left third molar (L-16) was the source of the subsequent trouble.

The day after extraction the gum in the vicinity of the empty sockets became swollen and painful. During the ensuing two and one-half months the infection spread. The soft tissues of the left side of the mandible were incised on three occasions, and the right mandibular region on one. Fever did not appear until 12 March: it was low grade, ranging between 99 and 101.5° F. Sulfadiazine had been given from the start, the blood level of the drug varying between 4.0 and 20 mg. per cent. The face and neck had been exposed to therapeutic doses of x-rays, staphylococcus toxoid had been administered, and several transfusions of blood given. On 12 April the face was still badly swollen, particularly over the left mandibular region. Trismus was pronounced. The parts of the mandible exposed by incision were superficially eroded. On 15 March a fenestrated tube was inserted into the left temporal fossa and brought out at the angle of the mandible. Through it the wound was irrigated with azochloramid. On 28 April the incision was deepened, and some holes were drilled into the mandible in an effort to afford better drainage. Even so, the infection continued to spread and occasionally it became necessary to incise newly formed pus pockets.

About the middle of May, purulent material began to discharge from the left ear. Neurologic examination at this time showed the right pupil larger than the left, but the finding could not be confirmed a few days later. On 6 June there was some ecchymosis of the palpebral conjunctiva of the left upper lid and the eyes seemed unduly prominent; there were also diplopia and nystagmus on left lateral gaze. On 8 June the left temporomandibular joint was drained of pus. Three days later a left mastoidectomy was performed; besides purulent mastoiditis a subjacent epidural abscess and thrombosis of the left lateral sinus were found. At about this time it became evident that the patient had an acute leptomeningitis. Death occurred on 12 June, 136 days after tooth extraction.

**Laboratory Data.**—Roentgenograms on 13 April revealed severe osteomyelitis of the left mandible, and on 7 June evidence of left mastoiditis. Cultures of pus from the left mandibular region on 5 March revealed nonhemolytic streptococci; and on 29 March, facultative nonhemolytic streptococci and staphylococci. Spinal fluid study on 12 June: WBC 1,000 (P 56, L 44). Throughout the illness the blood showed persistent leucocytosis. Post-mortem culture of spinal fluid and heart blood yielded alpha hemolytic streptococcus and hemolytic *Staphylococcus albus*.

**Pathology.**—Examination of the skull at autopsy disclosed on the left an osteomyelitis of the mandible, temporomandibular joint, and adjoining zygomatic process, the more lateral portion of the greater wing of the sphenoid, the squamous and petrous parts of the temporal bone, and the mastoid. On removal of the drain from the left mastoid considerable epidural pus was exposed. The lateral sinus contained an organizing thrombus 2.0 cm. in length (Fig. 26). The right middle ear and mastoid were free from change, as were also the paranasal sinuses.

A leptomeningeal exudate covered the base of the brain and the left hemisphere of the cerebellum. Microscopic examination disclosed suppuration of

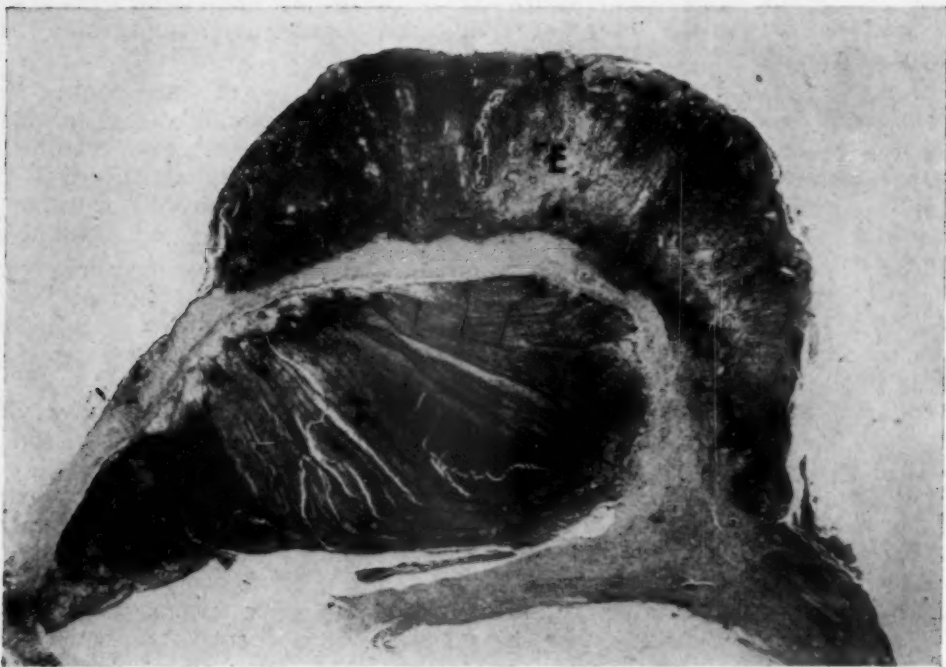


Fig. 26.—Thrombosis of the left lateral sinus. A fibrinopurulent thrombus (*T*) distends the sinus. Epidurally (*E*) there is a collection of pus adherent to the sinus wall. AMM Neg. 84324. Hematoxylin and eosin stain.  $\times 35$ .

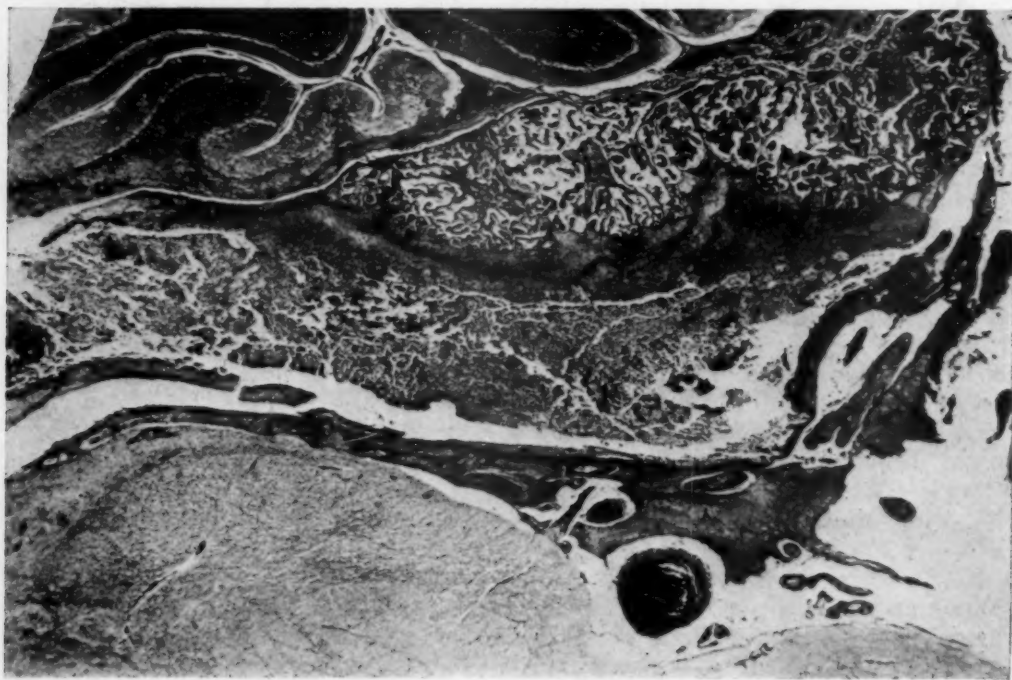


Fig. 27.—Purulent exudate within the fourth ventricle. Lying between the pons and cerebellum and infiltrating the surface of the choroid plexus is a heavy purulent exudate which doubtless extended into the fourth ventricle from the neighboring leptomeninges. AMM Neg. 84316. Hematoxylin and eosin stain.  $\times 35$ .

the choroid plexus of the fourth ventricle and of the adjacent ependyma (Fig. 27). The leptomeningeal exudate extended deeply into the cerebellum via perivascular spaces but no abscess was in evidence.

*Comment.*—This is an instance of gradual spread of osteomyelitis, ultimately involving the temporal bone and leading to thrombosis of the lateral sinus. Death followed extension to the leptomeninges.

CASE 26 (AMM Accession 33281).—Male, colored, age 33. *Cellulitis of the maxillary region, right; abscess of lungs; abscesses of maxillary, pterygoid, subtemporal, and postauricular regions, right; osteomyelitis of the sella turcica; purulent sphenoidal sinusitis, left; abscess of the orbits, first right and then left; purulent sinus thrombosis (lateral), bilateral; abscess of the cerebellum, right, with leptomeningitis of the basal cisternae.* Death occurred 28 days after tooth extraction.

*Clinical Data.*—On 20 May, 1930, the lower right first molar (R-14) was extracted, and on 21 May the upper right first bicuspid (R-4). The reason for extraction and the type of anesthesia employed are not known. The subsequent trouble started from the region of R-4.

On 22 May the right side of the face became swollen and painful, and soon the right temporal region became similarly affected. Chills and fever developed. When the patient entered hospital on 27 May the temperature was 101° F., the pulse 90. The swelling had spread to involve the right eyelids. Cervical lymph nodes were moderately enlarged. The temperature and pulse continued to rise. On 30 May, multiple incisions were made in the right cheek and adjoining temporal and postauricular regions, and copious pus evacuated. The wounds were irrigated repeatedly with Dakin's solution.

On 4 June the left eyelids began to swell and two days later became fluctuant, whereupon the upper lid was incised and the orbit drained. Both eyes, particularly the right, were unduly prominent, and their conjunctivae edematous. Toward the end, the cellulitis spread down into the right supraclavicular region. Death occurred on 18 June, 28 days after extraction.

*Laboratory Data.*—Culture from the right maxillary abscess on 30 May yielded *Staphylococcus aureus*. Blood leucocytes averaged 30,000 (P 90, L 10). Blood culture (date not given), negative.

*Pathology.*—Examination of the floor of the skull revealed erosion of the sella turcica. The left sphenoidal sinus was coated with purulent exudate. Both orbits contained pus. On the right side the orbital abscess was continuous through the inferior orbital fissure with the abscess of the pterygoid fossa. (The condition of the bones of the orbits was not described.) Exploration of the venous sinuses disclosed soft purulent thrombi in both lateral sinuses.

The leptomeninges, especially those of the basal cisternae, were laden with purulent exudate. Sections of the brain revealed in the right lobe of the cerebellum a small superficial abscess which communicated freely with the leptomeninges.

The lungs contained numerous abscesses, most of them well encapsulated.

*Comment.*—The abscess of the right orbit occurring early in the course of the illness probably was the result of extension of the subtemporal infection through the inferior orbital fissure. The development of the left orbital abscess, which occurred one week later, may have been due to spread through the lateral wall of the sphenoidal sinus.

That the intracranial lesions were hematogenous seems doubtful even though metastatic abscesses had become established in the lungs. The cerebellar abscess and cisternal leptomeningitis were a result of spread from the infected



right lateral sinus, but the source of the sinus infection is not evident. The infective organism doubtless reached the lateral sinuses by way of tributaries: for instance, the emissary veins traversing the mastoid foramen, draining the postauricular region (in this case, the seat of suppuration), and the superior petrosal sinuses draining more anterior parts of the skull (where osteomyelitis was severe). That lateral sinus thrombosis may complicate infections of the scalp is recognized (Byers and Hass). No information on the condition of the temporal bones is available.

CASE 27 (AMM Accession 36198).—Male, Filipino, age 31. *Cellulitis of the mandibular region followed by submandibular abscess, right; sinus thrombosis (lateral), bilateral.* Death occurred 10 days after tooth extraction.

*Clinical Data.*—On 15 June, 1931, owing to "pain in the right lower jaw" of several days' standing, the lower right second molar (R-15) was extracted. Data on the type of anesthesia employed are not available. After extraction the pain persisted, and soon the tissues at the angle of the right jaw became swollen.

The patient was admitted to the hospital on 20 June. His temperature was 101.8° F., the pulse 120. The anterior cervical lymph nodes of the right side were somewhat enlarged. On 22 June an abscess in the region of the right mandible was incised and drained. No other information is available. Death occurred 25 June, 10 days after tooth extraction.

*Laboratory Data.*—Blood examination on 22 June: WBC 15,700 (P 84, L 16). Urinalyses, Wassermann and Kahn, and blood culture on 22 June were all negative.

*Pathology.*—Examination of thoracic and abdominal contents failed to reveal anything of note. Both lateral sinuses were filled with well-organized thrombi; other intracranial sinuses were empty. The leptomeninges contained a "gelatinous exudate." Section of the brain showed nothing of significance.

*Comment.*—The cause of death is not evident. From the rapid advance of the cellulitis of the jaw and from the fulminant clinical course one may assume that bacteremia existed. If this be true, one may conjecture that the sinus thrombosis was the result of bacteremia.

Combined lateral and sigmoid sinus thrombosis, bilateral in distribution, has been noted also by Móczár in a case of purulent cellulitis originating in the region of the lower left third molar (L-16) and spreading to the left temporal region, but the pathogenesis of the thrombosis was not clarified.

#### TRANSVERSE MYELITIS

One case of transverse myelitis concludes the series of complications of tooth extraction. The inference that the myelitis was actually the consequence of the extraction is based chiefly on bacteriologic evidence.

CASE 28 (AMM Accession 106817).—Male, white, age 31. *Edema of the pharynx; interstitial pneumonitis, bronchopneumonia, and beginning pulmonary abscesses; transverse myelitis.* Death occurred 11 days after tooth extraction.

*Clinical Data.*—On 11 December, 1943, because of periapical abscess, the upper left first bicuspid (L-4) was extracted. Local procaine anesthesia was employed. There had been no significant previous illnesses. After the extraction the patient had considerable local discomfort, and because of "feverishness" spent the day in bed. The next evening, after putting in a full day of work, he again felt feverish and had a chill.



On 13 December the patient was admitted to hospital. The temperature was 104.6° F., the pulse 90. The gum in the region of the extracted tooth was not unduly swollen. The pharynx was reddened and slightly edematous. Examination of the chest, abdomen, and central nervous system was negative. On 15 December, after a temporary fall, the temperature rose to its previous height. On the same day there was clinical and roentgenologic evidence of patchy consolidation of the upper lobe of the left lung. A diagnosis of "atypical pneumonia" was made. By 17 December the lower lobe of the left lung was similarly affected. On 18 December the use of sulfadiazine was instituted, the blood level on 20 December being 4.8 mg. per cent.

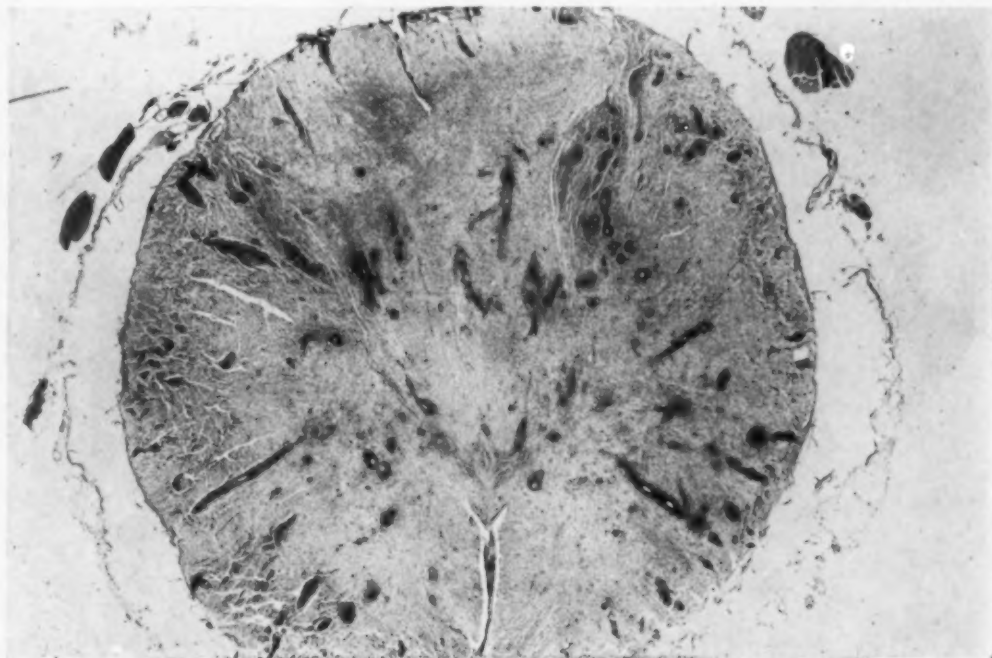


Fig. 28.—Transverse myelitis of lower cervical region. There are myriad perivascular abscesses in both white and gray matter. These are shown at higher magnification in Fig. 29. The leptomeninges are relatively normal except in the region of the anterior median fissure where there is an infiltrate of inflammatory cells. AMM Neg. 78727. Hematoxylin and eosin stain.  $\times 10$ .

On 20 December, after preliminary abdominal pain, the patient noticed that he could not move his lower extremities. He felt numb up to the waist, and could not urinate. By 22 December the paralysis had advanced to involve the upper extremities. Sensibility below the level of the fourth thoracic segment had been lost. Neither deep nor superficial reflexes could be elicited. The cervical lymph nodes, especially those on the right, were somewhat enlarged. Respirations became labored and "wheezy." There was evidence of a complete consolidation of the left lung. Abdominal distention increased, and there was fecal incontinence. Later in the day the neck was found to be stiff. Spinal tap revealed meningitis. Death occurred on 22 December, 11 days after tooth extraction.

**Laboratory Data.**—Blood examination on 13 December, WBC 9,600, and on 20 December, WBC 10,600 (P 74, L 26). Spinal fluid examination on 22 December, WBC 2,300 (P 97, L 3). Cultures of socket of extracted tooth: non-hemolytic *Staphylococcus aureus* (coagulase positive) and nonhemolytic gamma streptococcus; of lung tissue, nonhemolytic *Staphylococcus aureus* (coagulase

positive); of cerebral meninges and heart blood, no growth. Bacterial stains of the spinal cord failed to disclose organisms.

*Pathology.*—In the lungs the most striking changes were in the left upper lobe. The parenchyma contained innumerable minute yellowish foci. Microscopically, many of these foci proved to be early abscesses; in addition, numerous alveolar walls were hyperplastic, and within alveolar sacs there were inflammatory cells mixed with fibrin, and in some regions fibroblasts. The bronchiolar walls, in places denuded of mucosa, were heavily invaded by lymphocytes and histiocytes. The picture was that of an interstitial pneumonitis with bronchopneumonia and early formation of multiple abscesses.

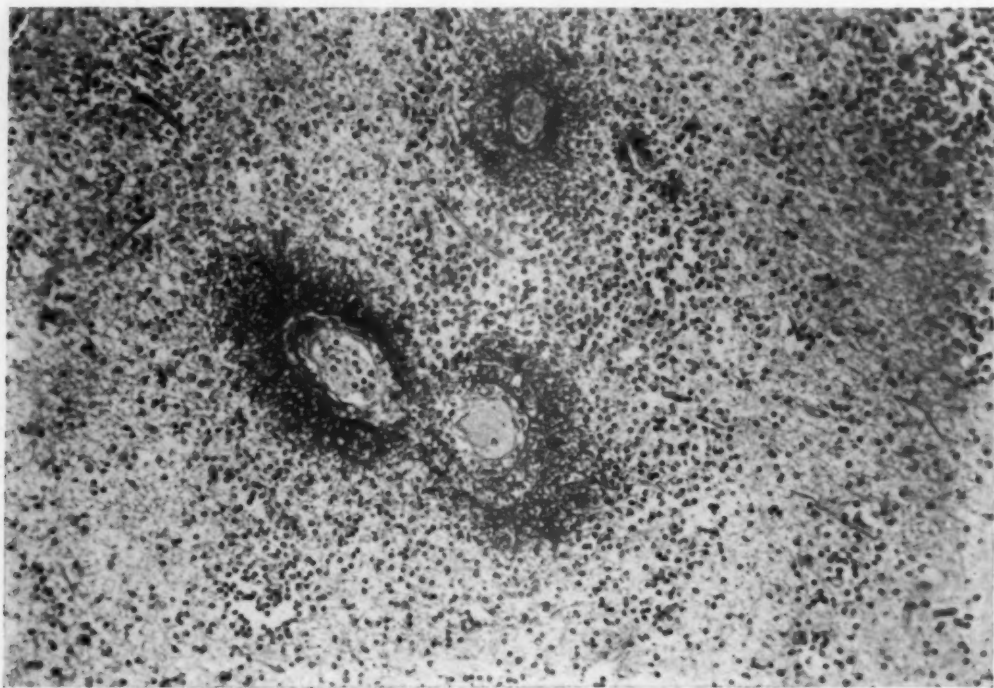


Fig. 29.—Perivascular abscesses in transverse myelitis. There is severe necrosis of the walls of the blood vessels. In the adjoining parenchyma there are fibrin deposits and infiltrates of polymorphonuclear leucocytes and histiocytes. AMM Neg. 78732. Hematoxylin and eosin stain.  $\times 200$ .

The spinal cord was severely involved in its lower cervical, thoracic and upper lumbar segments (Fig. 28). The white matter was most affected. Virtually all the blood vessels had undergone necrosis. An occasional vessel contained a hyaline thrombus. In the adjacent extra-adventitial parenchyma there were fibrin deposits and widespread heavy infiltrates of polymorphonuclear leucocytes and histiocytes (Fig. 29). The over-all picture was that of myriad small perivascular abscesses. Special stains failed to reveal organisms. The leptomeninges contained a few lymphocytes. The dura was normal. The spinal roots of the mid-thoracic region were hyperemic but contained no inflammatory cells. The brain and its meninges, the venous sinuses, the paranasal sinuses, and the petrous portion of the temporal bones were without change.

*Comment.*—The presence of a pathogenic form of staphylococcus in the empty tooth socket and in the lung, as disclosed by culture, suggests that the pulmonary abscesses were metastatic. The subsequent myelitis was apparently a further complication.

## DISCUSSION

The foregoing case histories illustrate the wide variety of intracranial complications which may ensue after tooth extraction. An over-all survey of the group (Table I) discloses direct extension of the infection to the intracranial cavity as the essential cause of death in 17 cases, and spread by way of the general circulation in the remaining 11. In some of the cases there was both direct and hematogenous spread. The discussion is introduced by a consideration of the relation of the various intracranial lesions to specific teeth extracted.

THE RELATION OF INTRACRANIAL COMPLICATIONS  
TO SPECIFIC TEETH EXTRACTED

Table I lists the teeth extracted and the chief complications in each of the 28 cases. Extractions from the upper jaw were numerically the same as those from the lower jaw, 14 in each. According to some observers, complications are more common in infections of the lower than of the upper jaw. Thus, in 24 cases of fatal complications of dental infection reported by Móczár, of which approximately 50 per cent are intracranial, the infections commenced in the lower jaw in 23 and in the upper jaw in 1. The figures of Childs and Courville, derived from a review of 59 cases of cavernous sinus thrombosis complicating dental infections, also show a predominance of lower jaw involvement: 36 as against 23. A sum total of all these cases reveals that fatal complications of dental infections of the lower jaw are approximately twice as frequent as those of the upper jaw.

Left versus right showed differences, there being 16 of the former and 11 of the latter (with 1 undetermined). Analysis of the series of Móczár, Childs and Courville, and our own shows a ratio of about 3 to 2 in favor of the left side. This difference may or may not prove fortuitous when more cases are available for analysis.

The number of teeth extracted varied from one to six. In 19 of the cases only one tooth was removed, a point which in itself would seem to indicate that the danger lies elsewhere than in multiple extraction. This view is borne out also by the incidence of bacteremia after single tooth extraction: of the 10 cases of spread of the infection via the general circulation to the intracranial cavity, and 1 to the spinal cord, there were 9 in which only one tooth was removed. In instances in which the greatest number of teeth were extracted (six teeth in Case 3, five in Cases 1, 16, and 15, and four in Case 2) there was no bacteremia.

Bacteremia is somewhat more frequent after extraction from the lower than from the upper jaw: 9 cases as against 5 (Table IV). The reason for this is not apparent, unless it be that drainage immediately after extraction is less adequate from the lower than from the upper jaw.

A point which quickly meets the eye in a survey of Table I is the predominance of posterior teeth extracted. Molars outweigh all the rest, the third molar in particular. No correlation could be found between the tooth extracted and the subsequent clinical course: of 8 cases in which only third molars were extracted there were 5 different types of intracranial complica-







4	0	L	+	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	From blood (post mortem): <i>Strep. hemolyt.</i>
8	0	R	+	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	From cervical abscess: same From blood (on 3 occasions): none
21	0	R	+	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	From left orbit (32nd day): diplococcus (gram-neg.)
24	0	L	+	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	No cultures
25	0	L	+	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	From blood and spinal fluid (post mortem): <i>Staph. alb. hemolyt.</i> , and <i>Strep. hemolyt.</i>
111	0	L	+	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	From brain abscess and sphenoid and ethmoid sinuses (post mortem): pneumococcus, not typed
18	0	L	+	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	No cultures
16	0	R	+	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	No cultures
27	0	L	0	+	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	From blood (7th day): none
17	0	L	0	+	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	From blood (17th day) and from submental abscess (post mortem): diplococcus, bacilli, and staph.
12	0	L	0	0	+	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	From empyema (12th day) and from lung abscesses (post mortem): beta hemolyt. strep.
15	0	L	0	0	+	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	From abscesses of orbit and brain (post mortem): staph., type unspecified
19	0	R	0	0	+	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	From blood (6th day and post mortem): <i>Strep. nonhemolyt.</i>
13	0	R	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	From right mandibular region (9th day): <i>Strep. virid.</i>

The symbols +, 0, and - are meant to connote the following: + indicates the teeth removed from either the upper or the lower jaw, or both, and that the pathologic or other findings are positive; 0, that teeth were not removed, or that the pathologic findings were negative; -, that no information on the point is available.

\*These extractions, done approximately at the same time as the others, were regarded as noncontributory to the ultimate outcome.

†Terminal bacteremia and terminal leptomeningitis are not included in this table.

‡In Cases 26, 18, and 19 there were metastatic abscesses in viscera but the spread to the intracranial cavity was not regarded as hematogenous.

tion. The tendency for infections in the vicinity of molar teeth to lead to intracranial complications doubtless is to be ascribed to anatomic relations: pus arising in the back of the jaw tends to collect between muscles of mastication and to spread rapidly upward in fascial planes, whereas that arising in the front of the jaw has free access to the oral cavity or to the exterior.

The figures show that extension of suppurative cellulitis to the base of the skull is more common after extractions of upper than of lower teeth: 10 cases versus 5. Advancing suppuration ensued in all the cases in which molars were extracted from the upper jaw, and in less than one-half of the cases after extraction of molars from the lower jaw.

A further point of interest is the relation of cavernous sinus thrombosis to teeth extracted: all nine cases of thrombosis occurred after the extraction of molars.

#### BACTERIOLOGIC FINDINGS

Cultural studies of bacterial flora are available in 21 of the 28 cases (Table I). A wide variety of organisms was disclosed in each of the categories of intracranial spread. Thus, in infections gaining the intracranial cavity by way of the general circulation there were streptococci, staphylococci, diplococci, and pneumococci, with *streptococci* most frequent (Table II), while in infections reaching the intracranial cavity by direct spread there was much the same flora, but with *staphylococci* in the foreground (Table III).

TABLE II

BACTERIOLOGIC DATA ON HEMATOGENOUS INFECTIONS FOLLOWING TOOTH EXTRACTION

CASE NUMBER	DAYS SURVIVAL AFTER EXTRACTION	ORGANISMS CULTURED	SITES FROM WHICH CULTURES WERE MADE	ESSENTIAL CAUSE OF DEATH
7	41	<i>Strep. virid.</i>	Brain abscess	Brain abscess
13	64	<i>Strep. virid.</i>	Right mandibular abscess	Brain abscess
9	23	<i>Strep.</i> (type unspecified)	Brain abscess	Brain abscess
4	5	<i>Strep. hemolyt.</i>	Heart blood	Leptomeningitis
12	22	<i>Strep., beta hemolyt.</i>	Pleural pus and lung abscesses	Brain abscesses
19	23	<i>Strep. nonhemolyt.</i>	Blood	Cav. sinus thrombosis and leptomeningitis
10	137	<i>Staph. aureus</i> , anaerobic	Brain abscess	Brain abscess
28	11	<i>Staph. aureus</i>	Tooth socket and lung abscesses	Transverse myelitis
21	36	Diplococcus (gram-neg.), (unidentified)	Left orbital abscess	Brain abscesses
11	49	Pneumococcus (not typed)	Ethmoid and sphenoid sinuses and brain abscess	Brain abscess

The organisms recovered in cases of thrombosis of the *cavernous sinus* were streptococci (viridans in two, beta hemolytic in one, and nonhemolytic in one—Cases 23, 22, 12, and 19 respectively), diplococci (Case 21) and multiple (Case 17); in those of thrombosis of the *lateral (or transverse) sinus* the responsible organisms were staphylococci and streptococci (Cases 26 and 25). These two organisms were predominant also in cases of brain abscess (Cases 2, 3, 6, 7, 9, 10, 11, 14, and 15). From these data it is not possible to correlate organism with duration of the illness or with the type of lesion produced.

TABLE III

BACTERIOLOGIC DATA ON INFECTIONS REACHING THE INTRACRANIAL CAVITY BY DIRECT SPREAD

CASE NUMBER	DAYS SURVIVAL AFTER EXTRACTION	ORGANISMS CULTURED	SITES FROM WHICH CULTURES WERE MADE	ESSENTIAL CAUSE OF DEATH
15	28	Staph. (type unspecified)	Orbital and brain abscesses	Brain abscess
20	12	Staph. (type unspecified)	Maxillary abscess	Cav. sinus thrombosis
26	28	Staph. aureus	Maxillary abscess	Lat. sinus thrombosis and cerebellar abscess
2	28	Staph. aureus	Alveolar abscess	Subdural empyema and brain abscess
3	129	Staph. hemolyt. and Strep. hemolyt.	Frontal sinus and brain abscess, respectively	Subdural empyema and brain abscess
5	27	Staph. hemolyt. and Strep. hemolyt.	Preauricular abscess and heart blood, respectively	Leptomeningitis
6	38	Anaerobic Staph. and Strep. (unspecified)	Brain abscess	Leptomeningitis and brain abscess
25	136	Staph. alb. hemolyt. and Strep. alpha hemolyt.	Spinal fluid and heart blood	Ependymitis and lat. sinus thrombosis
14	28	Strep. nonhemolyt. and Staph. aur. hemolyt.	Maxillary abscess (brain abscess, no growth)	Brain abscess
22	48	Strep. virid.	Heart blood, maxillary abscess, and leptomeninges	Cav. sinus thrombosis and leptomeningitis
23	28	Strep. virid.	Heart blood, urine, and maxillary abscess	Cav. sinus thrombosis and leptomeningitis
16	15	Strep. (type unspecified)	Brain abscess	Brain abscess
17	20	Diplococcus, Strep. and Bacilli (unspecified)	Blood and submental abscess	Cav. sinus thrombosis

## HEMATOGENOUS INFECTIONS COMPLICATING TOOTH EXTRACTION

In 14 cases, or 50 per cent of the series, there was evidence of bacteremia which was either clear-cut or circumstantial (Table I). In three of these (Cases 26, 18, and 19) the organisms were disseminated only in the viscera, the intracranial lesions being the result of an independent spread of the infectious process through the base of the skull.\* Considering all 14 cases, it will be noted that 9 of the extractions were from the mandible, and 5 from the maxilla (Table IV). Single teeth were extracted in all except three (Cases 10, 11, and 18). It is of interest in infections borne by the blood stream that, excepting Case 10, the only teeth removed from the maxilla were *bicuspid*s and that, except for Case 13, all those extracted from the mandible were *molars*. The mouth was clean prior to extraction in the majority (approximately 11 of the 14). The teeth were removed for various reasons: periapical abscess, 3; local chronic osteomyelitis, 1; "pain in the jaw," 1; impaction, 1; "toothache," 1; malposition, 1; and not specified, 6.

A review of the circumstances of each of the 14 cases reveals that bacteremia occurred at varying intervals after extraction. Bacteremia is judged to have occurred (1) as the immediate result of extraction in seven (Cases 9,

\*Similar cases have been reported. In the case of Dixon, for instance, the patient had had the lower left first and second molars (L-14, 15) extracted, and the sockets curetted. Proptosis of the left eye, which ensued on the fifth day after extraction, was taken as evidence of thrombosis of the cavernous sinus, but two days later the proptosis began to recede, and then disappeared. At autopsy there were widespread metastatic abscesses in the viscera; the only intracranial lesion consisted of liquified thrombus within the left cavernous sinus.

7, 26, 28, 10, 12, and 19), (2) shortly after extraction in association with fulminant cellulitis of the jaw in two (Cases 4 and 27), (3) approximately one month after extraction as the result of surgical intervention in two (Cases 8 and 11), and (4) at undetermined times, probably early after extraction in two (Cases 21 and 18) and late in one (Case 13).

TABLE IV  
TOOTH EXTRACTIONS FOLLOWED BY HEMATOGENOUS SPREAD OF THE INFECTION

CASE NUMBER	FROM MANDIBLE	CASE NUMBER	FROM MAXILLA
4	Third molar	9	Second bicuspid
8	Third molar	7	First bicuspid
21	Third molar	28	First bicuspid
11	Third and second molars	26*	First bicuspid
18*	Third, second, and first molars	10	Lateral incisors (2)
27	Second molar		
12	First molar		
19*	First molar		
13	Central incisor		

\*In these 3 cases there was hematogenous spread only to viscera. The intracranial lesions in these cases were the result of direct spread through the base of the skull.

Bacteremia as the immediate result of extraction is relatively frequent (Richards; Okell and Elliott; Palmer and Kempf). After routine extractions, the organisms (usually *Streptococcus viridans*) may be cultured from the blood stream during the period of from ten minutes to one hour after extraction. In the vast majority of instances the bacteremia proves inconsequential. The cases reported here represent exceptions to the rule.

Bacteremia resulting from tooth extraction more frequently causes sub-acute bacterial endocarditis, but only when there has been previous damage to heart valves (Abrahamson; Brown; Clagett and Smith; Geiger). In this series of 14 cases of bacteremia there were no instances of endocarditis; nor was there evidence of previous cardiac disease except for old myocardial infarcts with myocardial aneurysm in one instance (Case 13).

*Brain abscess* constituted by far the most frequent intracranial complication in cases in which bacteremia, either proved or circumstantial, was a feature. Of the 11 cases in which there was evidence of bacteremia, brain abscess was present in 7.\* The cases fall into two categories: those characterized by single brain abscess and those by multiple ones. The *single brain abscesses* were of frontoparietal location in three instances (Cases 9, 10, and 11) and temporal in one (Case 7). All were well encapsulated and could have been effectively drained surgically. The outcome would have been favored by the absence of metastatic abscesses elsewhere in the body. Three of the brain abscesses were contralateral to the site of tooth extraction; in the fourth (Case 10) it is impossible to say whether the abscess was contralateral or ipsilateral because teeth were extracted from both the right and the left sides. In none of the cases were there complications at the site of extraction except for postmandibular cellulitis in one (Case 11).

The initial symptoms of brain abscess in each of the cases were as follows: In Case 9 (*frontoparietal abscess*) the first untoward symptom was that of convulsive seizure, followed in rapid succession by nine more; the convulsions oc-

\*To this series of 7 may be added an instance of blood-borne suppurative encephalitis with rapid extension of the infection to the ventricular system (Case 8).



curred on the tenth day, and death on the twenty-third day after extraction. In Case 11 (*frontoparietal abscess*) the patient was recovering from postmandibular cellulitis, for which a tooth had been extracted to afford drainage, when on the thirteenth day after the secondary extraction, he had a sudden convulsive seizure followed by three others; he died twelve days later. In Case 10 (*frontoparietal abscess*) the patient, after being well for several weeks after extraction, rapidly became disoriented and developed slurring of speech and hemiparesis; death occurred four days later. In Case 7 (*temporal lobe abscess*) the symptoms of involvement of the brain occurred shortly after extraction: severe headache on the next day and hemiparesis on the third day. Meningitis ensued but was brought under control by sulfadiazine. All was going well when, on the thirty-seventh day, headaches recurred and complete motor aphasia developed. At operation the suspected brain abscess was not located. Death occurred on the forty-first day after extraction.

*Multiple brain abscesses* of hematogenous origin, of which there were three (Cases 21, 12, and 13), were all associated with metastatic abscesses of the lungs, and, in one case, abscess of the right kidney as well. In all three a purulent cellulitis of the jaw followed extraction. In two instances (Cases 21 and 12) there was rapid development of stupor after intracranial involvement, whereas in the other (Case 13) the data are too inadequate to reconstruct the sequence of clinical events.

The other 4 of the 11 cases of hematogenous spread of the infection after tooth extraction fall into the following categories: leptomeningitis (Case 4), suppurative encephalitis and ependymitis (Case 8), lateral sinus thrombosis (Case 27), and transverse myelitis (Case 28).

#### INTRACRANIAL COMPLICATIONS AS A RESULT OF DIRECT SPREAD OF THE INFECTION THROUGH THE SKULL

Direct spread of infection from the site of extracted teeth to the intracranial cavity usually occurs as follows: One or more teeth are extracted, regional suppurative cellulitis ensues, the pus burrows along fascial planes to the base of the skull, and en route it may invade paranasal sinuses and an orbit; sooner or later the cranial wall is penetrated either by means of veins, leading to sinus thrombosis, or via the bone itself, causing subdural empyema, leptomeningitis, or brain abscess, singly or combined. The extracranial collection of pus may be so deeply situated that it is either not recognized or not reached by surgical means.

Cases of direct spread of infection through the skull numbered 17; two other cases (Cases 12 and 21) belong to this group even though death was due to hematogenous brain abscesses (Table V). Considering the 19 cases, one finds that instances of upper and of lower jaw extractions are numerically about the same: 10 from the upper, 9 from the lower. Ten of the extractions were from the left side, and the nine from the right. Molars were removed in all save one (Case 26).

*Involvement of Paranasal Sinuses.*—The incidence of suppurative sinusitis in this series could not be determined with precision because of the lack of information in some of the protocols. One gains the impression that the incidence is higher than the available data indicate. The sphenoidal sinus alone was

TABLE V

DATA ON CASES OF SPREAD OF INFECTION INTO THE INTRACRANIAL CAVITY VIA OSSEOUS AND VENOUS ROUTES  
(Twelve Cases of Osseous Spread Are Listed Above the Center Line, and Seven of Venous Spread Below This Line)

CASE NUMBER	TEETH EXTRACTED*		DAYS SURVIVAL AFTER EXTRACTION	SITES OF OSTEOMYELITIS†										SINUSITIS				CHIEF INTRACRANIAL LESIONS						ORGANISMS CULTURED		
	UPPER	LOWER		MANDIBLE	MAXILLA	FRONTAL	SPHENOID (GREATER WING)	SELLA TURCICA	SQUAMOUS	TEMPORAL	PETROUS	MASTOID	INTRAORBITAL ABSCESS	MAXILLARY	FRONTAL	ETHMOIDAL	SPHENOIDAL	SUBDURAL EMPYEMA	LEPTOMENINGITIS	SINUS THROMBOSIS	FRONTAL	TEMPORAL	MULTIPLE	CEREBELLAR	EXTRA-CRANIAL	INTRA-CRANIAL
1	+	0	2d bicuspid, 1st, 2d, 3d molars (L-5, 6, 7, 8)	0	-	0	+	+	+	-	0	0	0	0	-	+	+	R, L	-	-	-	-	-	-	0	0
2	+	0	1st, 2d, 3d molars (R-6, 8, 8)	-	-	-	+	+	-	-	0	-	-	-	-	-	-	+	-	-	-	+	-	-	Staph. aureus	-
3	+	0	Cuspid, bi-cuspid, molars (L-3, 4, 5, 6, 7, 8)	+	+	+	0	0	0	0	0	+	+	+	-	-	0	+	-	-	+	-	-	Staph. hemolyt.	Strep. hemolyt.	
5	+	0	3d molar (R-8)	+	-	-	+	-	-	0	0	-	+	+	-	-	+	-	+	-	-	-	-	Staph. hemolyt. & Strep. hemolyt.	-	
6	+	0	3d molar (R-8)	0	0	0	+	-	0	0	0	0	0	0	0	0	+	-	+	-	-	+	-	Staph. aureus	Staph. & Strep. (un-specif'd)	
14	+	0	1st molar (L-6)	-	+	0	0	-	0	0	0	+	-	-	-	-	-	-	-	-	+	+	-	Strep. nonhemolyt. & Staph. aur. hemolyt.	0	



purulent in five instances (Cases 1, 6, 19, 21, and 26), the maxillary and sphenoidal in one (Case 5), the maxillary and frontal in one (Case 3), and all sinuses in one (Case 22).

The *maxillary sinus* is liable to infection when, during removal of a tooth, a communication is created between the sinus and the mouth. That maxillary sinusitis is relatively common after extraction is understandable from anatomic relations. "The teeth whose roots are in relation to the floor [of the maxillary sinus] vary from the molars as a minimum to the molars, premolars, and the canine as a maximum. The roots of the first two molars produce eminences in the floor, and, though usually covered with thin bone, they may perforate the floor." (Cunningham.) The roots of three molars and one bicuspid as they relate to the maxillary sinus are shown in Fig. 30. Occasionally a prolapse of the mucous membrane of the maxillary sinus into the empty tooth socket after extraction constitutes a warning signal that maxillary sinusitis may ensue. In this series there were two instances of maxillary sinusitis following tooth extraction.

In one instance (Case 3), in which multiple teeth were removed (L-3, 4, 5, 6, 7, and 8), the patient soon noticed that with respirations he could feel air going out of the cheek bone. Osteomyelitis of the maxillary sinus ensued; the infection reached the orbit, from which it spread to the frontal bone and sinus, gaining entrance into the subdural space of the anterior cranial fossa and ultimately the brain. In the other instance (Case 5), in which the upper right third molar was removed, there was preliminary maxillary sinusitis accompanied by severe osteomyelitis of the sphenoidal sinus and the adjacent greater wing of the sphenoid and, in time, acute leptomeningitis. At autopsy a probe could be passed through the socket of the removed tooth into the maxillary sinus.

The *sphenoidal sinus* was the one most frequently involved. Seven instances (Cases 1, 5, 6, 19, 21, 22, and 26) are to be found in the autopsy protocols. In most of these cases the adjacent greater wing of the sphenoid was the seat of osteomyelitis, suggesting that the sinusitis was incidental to osseous spread of the infection. In two instances (Cases 19 and 21) in which the greater wing of the sphenoid was spared, the sphenoidal sinusitis may have been secondary to cavernous sinus thrombosis.

On occasion the sphenoidal sinus may erode to such a degree that its purulent contents may empty into the nostril when the patient blows his nose or when the sinus is irrigated. Such a case has been described by Lodge. The patient had purulent cellulitis in the region of the upper second and third molars and subsequently an extension of the infection to the bone of the skull, leading to necrosis of the cavum sellae, thrombosis of the cavernous and superior longitudinal sinuses, and of ophthalmic veins, together with local subdural abscess of the middle cranial fossa and leptomeningitis of the anterior cranial fossa. The clinical course extended over fifty-three days, the nasal discharge beginning on the fortieth.

The *ethmoidal sinuses* did not figure in the complications of tooth extractions in this series, probably because they were so seldom explored at autopsy. Owing to their close anatomic relations, the posterior cells of the ethmoidal



sinuses and the sphenoidal sinus are often jointly involved. In only one instance (Case 22) was suppurative ethmoidal sinusitis disclosed, and then at autopsy.

Involvement of the ethmoidal sinuses as a complication of tooth extraction is known to occur. As an example may be cited a case of Eagleton (his Case 7) in which death followed thrombosis of the cavernous sinus.

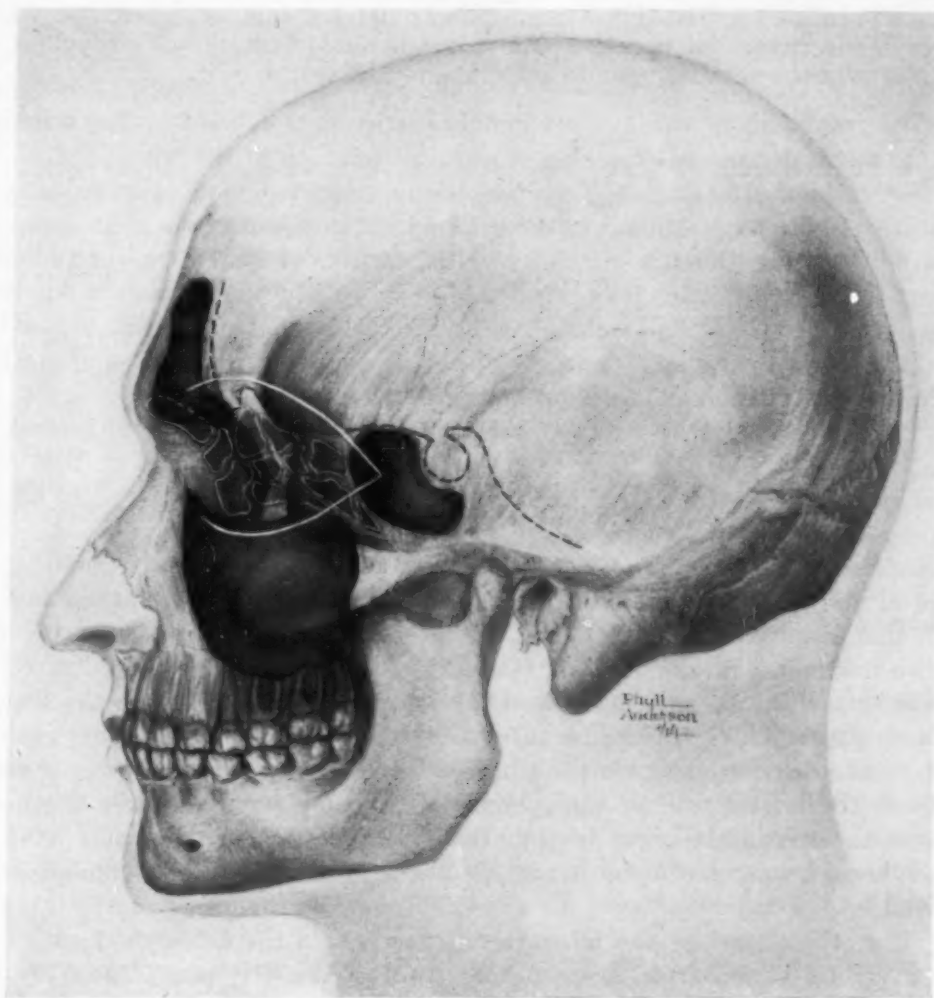


Fig. 30.—The relations of the paranasal sinuses of the left side of the skull. The mid-line of the base of the skull is indicated by the broken dark line, and the outline of the orbit by the white line. The frontal, maxillary, and sphenoidal sinuses are in dark tone, and the ethmoidal sinuses in light. The duct of the frontal sinus lies in close relation to the anterior ethmoidal cells. The anterolateral aspect of the sphenoidal sinus is medial to the apex of the orbit, and is contiguous with posterior ethmoidal cells. The upper part of the maxillary sinus is adjacent to the orbit, while the floor is closely related to the roots of the molars and the second bicuspid, and sometimes those of other teeth. The division of the ethmoidal sinuses into two parts, anterior and posterior, is indicated. After tooth extraction the sphenoidal sinus is the one most frequently involved by ascending infections. AMM Neg. 83847.

The patient, a young child, had extensive suppuration of an upper right molar and swelling of the eyelid and face. The tooth was extracted. Shortly thereafter the right orbit became abscessed, and on the fifth day after extraction it was incised and drained. Three days later there was swelling and venous engorgement over the right temporal, parotid, and zygomatic regions.

Death occurred on the twelfth day after extraction. Removal of the roof of the right orbit at autopsy revealed free pus at the apex of the orbit as well as in the adjoining posterior ethmoidal cells. In the floor of the orbit there was a perforation which linked the orbit with the maxillary sinus. Both cavernous sinuses contained free pus.

Eagleton expressed the opinion that the sequence of events was as follows: thrombophlebitis originating in the region of the extracted tooth spread upward, causing necrosis of the walls of the maxillary and ethmoidal sinuses and suppuration of the orbital tissues, and extended further via orbital veins to reach the cavernous sinuses. The point is made that the osteomyelitis in this case was the result of venous infection, not the cause of it.

The *frontal sinus* was involved in only one instance (Case 3). The sinusitis was the result of upward extension of purulent cellulitis of the orbit.

*Intraorbital Abscess.*—Alveolar infections associated with progressive suppurative cellulitis may spread to the orbit by various routes, the most common, apparently, being through the infraorbital fissure. From upper cuspid and bicuspid teeth an infection may gain the orbit subperiosteally (Thoma). Another route is through the lateral walls of the sphenoidal and ethmoidal sinuses. Both are in relation to the medial aspect of the orbit—the sphenoidal sinus to the apical part of the orbit and to the optic nerve in the optic foramen.

There were, in all, 6 cases of intraorbital abscess (Table V). In two of them (Cases 15 and 21) the extractions were from the lower jaw (first and third molars, respectively) and in four (Cases 3, 14, 20, and 26) from the upper jaw (from Case 3, the cuspids, bicuspid, and molars; from Case 14, the first molar; from Case 20, the third molar; from Case 26, the first bicuspid). In some of the cases the intraorbital pus was found at autopsy to be continuous with that situated behind the orbit. Bilateral orbital abscesses were present in two instances; in one of them (Case 21) the orbits were involved in rapid succession, while in the other (Case 26) a week intervened before the second became purulent. Spread of an infection from one orbit to the other is difficult to account for except on the basis of extension of the infective process through the lateral wall of the sphenoidal sinus, or subperiosteally from one side of the sphenoidal sinus to the other. In both cases the greater wing of the sphenoid was osteomyelitic and, in the second case, the sphenoidal sinus as well.

The role played by the intraorbital abscesses in the subsequent course of the infection varied from case to case. In 2 of the 6 (Cases 14 and 15) the intraorbital involvement appeared to be of little consequence inasmuch as the infective process spread through the greater wing of the sphenoid, leading to the formation of temporal lobe abscesses. Also in Case 26 the intraorbital abscesses seem to have had little bearing on the subsequently developing lateral sinus thrombosis and cerebellar abscess. In the other 3 instances the intraorbital abscesses were apparently a determining factor in the ultimate outcome. Thus, in Case 3, the pus in the orbit spread upward to involve the frontal bone and frontal sinus and ultimately gained entrance into the subdural space of the anterior cranial fossa, while in Cases 20 and 21 cavernous sinus thrombosis ensued.

*Involvement of the Gasserian Ganglion and Divisions of the Trigeminal Nerve.*—In the literature on complications of tooth extraction is to be found an

occasional instance of gasserian ganglionitis which was regarded as due to upward spread of infection by way of trigeminal (perineural) sheaths from purulent foci of the jaw. Such a case, described by Koepf, Rosedale and Learn, is referred to in this discussion under the heading, *Sinus Thrombosis*: the infection traveled presumably via the sheath of the mandibular nerve. In a case recorded by Buckley, the infection following extraction of the upper first and second molars (R-14, 15) is said to have extended upward along the right maxillary nerve to the gasserian ganglion, then to the capsule of the hypophysis and to the right temporal lobe. In still another instance, that described by Bannes, the passage of the infective process along the sheath of mandibular nerve was clearly demonstrated.

The patient, aged 27, had had a bicuspid and two molars (L-12, 14, 16) extracted from the left mandible. Block anesthesia with novocain was employed. The patient was admitted to hospital eight weeks after extraction. There had been recurrent severe pains over the left side of the face and during the week before admission, trismus and frequent vomiting. A slight ptosis of the left upper eyelid was noted. The entire left side of the head and face was unduly sensitive to pressure and other stimuli, except for the lips and mandibular gums which were hypesthetic. Operation eleven weeks after extraction disclosed a moderate-sized abscess of the antero-inferior aspect of the left temporal lobe. Two days later the patient died. Examination of the brain at autopsy revealed temporal lobe abscess. On section of the mandibular and maxillary divisions of the left trigeminal, as they traverse the foramen ovale and rotundum, respectively, large beads of pus escaped from beneath the perineural sheaths. Pus was present also around and within the gasserian ganglion but not in the cavernous sinus, or elsewhere except in the temporal lobe. The infection was regarded as a complication of the mandibular block anesthesia, and it was concluded that the infection traveled to the brain by way of the sheath of the mandibular nerve.

Evidence of transmission of infection along divisions of the trigeminal nerve in this series of cases is scant. The clinical story in Case 2 suggests early extension of the infective process to the ophthalmic nerve: on the second day after extraction of upper molars the patient complained of tenderness of the right eye, and on the next day a ptosis of the right upper eyelid was observed. At autopsy on the twenty-eighth day after extraction gasserian ganglionitis was found. There was also a temporal lobe abscess and subdural empyema of the middle cranial fossa.

*The Routes of Penetration of the Skull by Infective Organisms Gaining the Intracranial Cavity.*—Infections complicating tooth extraction traversed the skull in 19 of the 28 cases. The essential route pursued was judged to be osseous in 12, and venous in 7 (Table V). In some cases both routes were followed.

Osteomyelitis of the greater wing of the sphenoid was one of the more striking pathologic changes in this series. It was present in eight of the cases. The region of the foramen ovale was the site most conspicuously involved (Fig. 31). Although direct spread of pus through the foramen ovale and rotundum has been described by Turner (in a case of cavernous sinus thrombosis and leptomeningitis after upward spread of infection from two abscessed lower molar) it was not observed in this series. In most of the cases of osteomyelitis of the greater wing of the sphenoid there was osteomyelitis of the sella turcica or purulent sphenoidal sinusitis, or both. The combined involvement in these instances



was regarded as part and parcel of the same invasive process. In most of the cases, either at operation or at autopsy, sometimes at both, a collection of pus was found at the base of the skull. Cavernous sinus thrombosis was regarded as a complication of osteomyelitis in two instances (Cases 22 and 23). After direct extension through the skull the infective process led to suppuration of various structures, as will be noted in Table V. Abscess of the leptomeninges was not encountered in this series, but has been recorded by Keegan and Ash in connection with erosion of the greater wing of the sphenoid following multiple extraction.

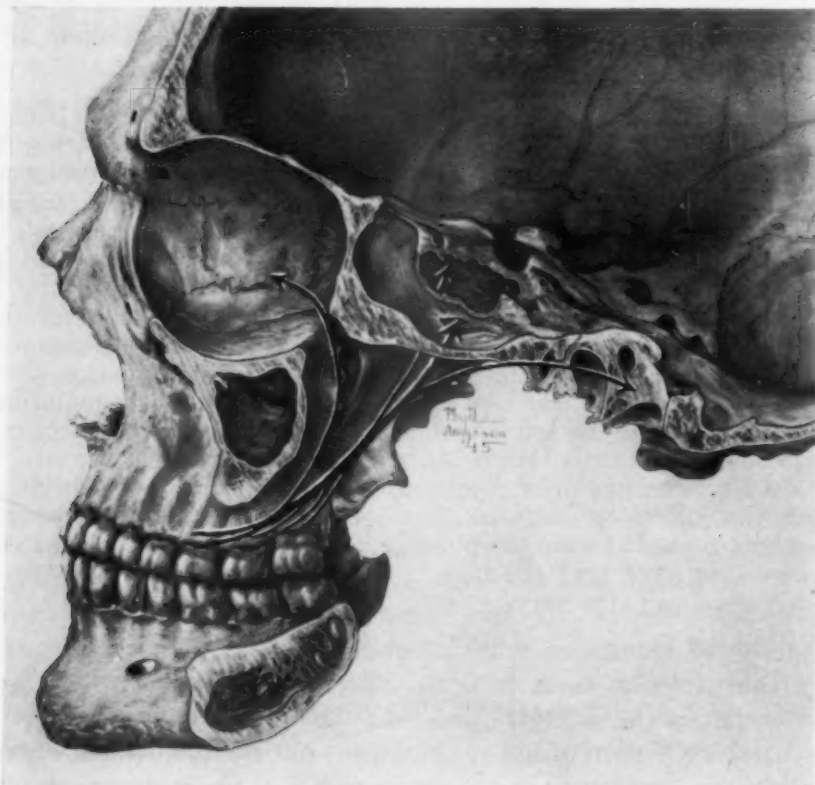


Fig. 31.—Pathways followed by infections which ascend from the jaw to reach the intracranial cavity. The most frequent site of penetration of the base of the skull by infections complicating tooth extraction is the greater wing of the sphenoid in the vicinity of the foramen ovale (indicated by the thicker arrow). The extracranial collection of pus is sometimes so deeply situated that its presence is not recognized clinically. The next most frequent cranial structures involved are the sphenoidal sinus and the overlying sella turcica. The infective organism may reach the sphenoidal sinus directly (as indicated by the arrow within the exposed sphenoidal sinus) or by contiguity from the greater wing of the sphenoid bone. (In some cases a sphenoidal sinusitis may be the result of retrograde thrombosis from a thrombosed cavernous sinus.) Another route of spread is through the petrous part of the temporal bone. In some cases the infective organism reaches the retrobulbar tissues in the manner indicated, and subsequently may enter the intracranial cavity via ophthalmic veins or by direct spread through orbital fissure or canal. AMM Neg. 84313.

The other essential means of spread was venous, which in all six cases resulted in thrombosis of the cavernous sinus. In two (Cases 20 and 21) the spread of the infection was doubtless through ophthalmic veins from orbital abscess. In the other four (Cases 17, 18, 19, and 24) the infection is believed to have spread upward from the pterygoid venous plexus.

*Intracranial Hemorrhage.*—Intracranial hemorrhage complicating infective sinus thrombosis does occur, although it has been regarded as rare by such competent observers as Lancial and Macewen. Byers and Hass noted subarachnoid hemorrhage in several instances of infective sinus thrombosis associated with



leptomeningitis, and Chisolm and Watkins have reported hemorrhages into the brain, leptomeninges, and spinal cord, in a case of cavernous sinus thrombosis and leptomeningitis following tooth extraction.

Intracranial hemorrhage was encountered in 5 of our cases: subdural in 1, and subarachnoid in 4. Three (Cases 19, 20, and 24) were associated with sinus thrombosis, one (Case 6) with temporal lobe abscess, and one (Case 5) with leptomeningitis.

The subdural hemorrhage (Case 24) was sufficiently massive to be a determining factor in the patient's death; after a stormy five-day clinical course marked by prostration, fear of impending death, and rapid development of peritonsillar abscess, the patient died, and at autopsy there were multiple sinus thrombosis, subdural hemorrhage over the base of the skull, and slight leptomeningeal hemorrhage.

Most of the subarachnoid hemorrhages were also abundant. They were present chiefly over the base of the brain and around the ventral aspect of the pons. In two (Cases 19 and 20) there was an associated cavernous sinus thrombosis, in one (Case 6) a brain abscess, and in one (Case 5) leptomeningitis as well as thrombosis of an ophthalmic artery.

The site of rupture of vessels in these cases was determined only in Case 5. That a subarachnoid hemorrhage may occur in cases of leptomeningitis is understandable from the frequency of suppurative phlebitis (Fig. 25), and its presence in sinus thrombosis can be explained on the basis of interference with blood flow in leptomeningeal veins.

*Brain Abscess.*—Data on brain abscess resulting from both direct and hematogenous spread of infections following tooth extraction are presented in Table VI. The cases of hematogenous brain abscess have been discussed on previous pages.

Brain abscess due to direct spread of the infection through the skull was encountered in 7 instances. In 5 of them the extractions were from the upper jaw, and in 2 from the lower jaw.

The temporal lobe was abscessed in 5 of the 7 (Cases 2, 6, 14, 15, and 16). Osteomyelitis of the underlying greater wing of the sphenoid was present in three of these (Cases 2, 6, and 15). In Case 16 a subacute inflammatory reaction of the hypophysial capsule and of the adjacent pachymeninx of the middle cranial fossa marked the area through which, presumably, the infective organism had passed.

Hardly could a more striking example of spread of an infection into the temporal lobe via the greater wing of the sphenoid be found than in Case 6: on attempting to remove the brain, the prosector encountered an adhesion between the dura and the inferior surface of the temporal lobe, and when he attempted to break it with his finger a copious amount of pus escaped from the abscessed temporal lobe; further examination revealed effective adhesion of dura and leptomeninges to the region through which presumably, the invading organism had passed.

In the remaining 2 of the 7 cases the abscesses were located in the frontal lobe and the cerebellum, respectively. The abscess confined to the frontal lobe\*

\*Abscess of the frontal lobe may occur as the result of spread of infection from a subtemporal abscess through the base of the skull in the region of the anterolateral aspect of the sphenoid bone. Such a case, ascribed to infection following mandibular block anesthesia, has been reported by Singer.

TABLE VI  
PATHOLOGIC AND BACTERIOLOGIC DATA IN THE CASES OF BRAIN ABSCESS

CASE NUMBER	TEETH EXTRACTED*		DAYS SURVIVAL AFTER EXTRACTION	NATURE OF SPREAD		SITES OF BRAIN ABSCESS†							OTHER INTRACRANIAL LESIONS							SITES OF METASTATIC ABSCESSES			ORGANISMS CULTURED	
	UPPER	LOWER		DIRECT	HEMATOGENOUS	FRONTAL	PARIETAL	FRONTO-PARIETAL	TEMPORAL	OCCIPITAL	ALL LOBES	CEREBELLUM	PACHYMEN-INGITIS	SUBDURAL EMPYEMA	ANTECEDENT LEPTOMENINGITIS	TERMINAL LEPTOMENINGITIS	SINUS THROMBOSIS	LUNG	PLEURA	KIDNEY	FROM BRAIN ABSCESS	FROM EXTRA-CRANIAL SOURCES		
2	+	0	1st, 2d, and 3d molars (R-6, 7, 8)	28	+	0	0	0	0	0	0	0	+	R, L	+	0	+	0	0	0	0	-	Staph. aureus	
3	+	0	Cuspid, bicuspids, and molars (L-3, 4, 5, 6, 7, 8)	129	+	0	0	0	0	0	0	0	+	+	0	+	0	0	0	0	0	Strep. hemolyt.	Staph. hemolyt.	
6	+	0	3d molar (R-8)	38	+	0	0	0	0	0	0	0	+	0	+	0	0	0	0	0	0	Anaerobic staph. & strep., un-spec'd	-	
14	+	0	1st molar (L-6)	28	+	0	0	0	0	0	0	0	+	0	0	+	0	0	0	0	0	Strep. non-hemolyt. & Staph. aur. hemolyt.	Staph. aureus	
26	+	0	1st bicuspid (R-4)	28	+	0	0	0	0	0	0	0	+	0	0	+	+	0	0	0	0	-	-	
15	0	+	1st molar (L-14)	28	+	0	0	0	0	0	0	0	+	0	0	0	0	0	0	0	0	Staph., type un-spec'd	Same	
16	0	+	Bicuspids and molars (R-12, 13, 14, 15, 16)	15	+	0	0	0	0	0	0	0	+	0	0	+	0	0	0	0	0	-	-	
7	+	0	1st bicuspid (L-4)	41	0	+	0	0	0	0	0	0	+	0	0	+	0	0	0	0	0	Strep. virid.	-	
9	+	0	2d bicuspid (L-5)	23	0	+	-	+	0	0	0	0	0	0	0	+	0	0	0	0	0	Strep., type un-spec'd	-	
10	+	0	Lateral incisors (L-2, R-2)	137	0	+	-	+	0	0	0	0	0	0	0	+	0	0	0	0	0	Staph. aur., anaerobic	-	
11	0	+	2d and 3d molars (L-15, 16)	49	0	+	-	+	0	0	0	0	0	0	0	0	0	0	0	0	0	Pneumococcus, not typed	Same	
12	0	+	1st molar (L-14)	22	0	+	-	-	-	-	+	0	0	0	0	+	+	+	+	+	0	Strep. beta hemolyt.	Strep. virid.	
13	0	+	Central incisor (R-9)	64	0	+	+	+	0	0	0	0	0	0	0	+	0	+	0	+	0	0	Diplococcus, unidentified	
21	0	+	3d molar (R-16)	36	0	+	R, L	+	0	0	0	0	+	0	0	0	+	+	+	0	0	0	0	

\*The sites of the extracted teeth indicated are the sources of origin of the spread of the infection. All the teeth removed in each case are listed in Table I.

†The sites of brain abscess are ipsilateral to tooth extraction unless otherwise specified.

(Case 3) was the end result of suppurative cellulitis which had spread to the orbit and thence to the frontal bone and frontal sinus. A tract leading from the outer surface of the dura to the brain abscess is illustrated in Fig. 8. In the other case (Case 26) an upper bicuspid was removed and the ensuing suppurative cellulitis spread to the base of the skull, extended into the orbits, and gained the scalp in the region behind the ear. The greater wing of the sphenoid was osteomyelitic. Either from the scalp or by way of the petrous bone (the study at autopsy does not indicate which) the infection reached the lateral sinus, producing a purulent thrombus and, in turn, an abscess of the cerebellum.

The temporal lobe abscesses were single in three instances (Cases 6, 15, and 16), triple in one (Case 2) and coextensive with the adjacent part of the frontal lobe in one (Case 14). Of the 5, one (Case 16) had ruptured into the lateral ventricle, another (Case 2) had extended as suppurative encephalitis to the threshold of the lateral ventricle, still another (Case 6) had remained imprisoned in the brain, while the remaining two (Cases 14 and 15) had seeped into the overlying leptomeninges. Some of the abscesses were relatively superficial; others deep.

A point deserving emphasis is that temporal lobe abscess may go unsuspected when leptomeningitis also is present. In two cases of this series, one of which was discussed under hematogenous brain abscess, the leptomeningitis was virtually cured by antibiotic drugs when brain abscess flared up. The circumstances in each of the cases are as follows:

In the one instance (Case 6) the patient developed leptomeningitis on the twenty-third day after tooth extraction. Given sulfadiazine, and later penicillin, he improved considerably and the leptomeningitis had all but disappeared on the twenty-eighth day when symptoms relating to the temporal lobe abscess developed. Death occurred on the thirty-eighth day after extraction. The other case (Case 7) is similar. Leptomeningitis became evident on the eighth day after extraction and under sulfadiazine therapy had virtually cleared on the thirty-seventh day when symptoms of cerebral involvement intervened. The patient died on the forty-first day after tooth extraction.

*Sinus Thrombosis.*—Thrombosis of dural sinuses occurred in twelve cases, or approximately 43 per cent of the series (Table VII). Unpaired sinuses (i.e., inferior and superior longitudinal,\* and straight) escaped involvement. Cavernous sinus thrombosis occurred in 9 cases, lateral sinus thrombosis in 3.

The cavernous sinus thromboses were somewhat more frequent after extractions of lower than of upper teeth: 6 as against 3. Molars alone were extracted in every instance except for one (Case 17) in which a second bicuspid as well was removed.

The circumstances preceding cavernous sinus thrombosis varied from case to case. Thus, in two (Cases 20 and 21) there were orbital abscesses which doubtless were the source of cavernous sinus infection, in another (Case 12) a postextraction abscess remained localized to the submaxillary region, in four (Cases 17, 18, 19, and 24) there was either abscess or nonsuppurative cellulitis

\*A survey of the literature on complications of tooth extraction disclosed thrombosis of the superior longitudinal sinus in two instances. One by Lodge, referred to in this discussion under the heading, *Sphenoidal Sinusitis*, was associated with leptomeningitis of the anterior cranial fossa. In the other, reported by Chisolm and Watkins, the superior longitudinal sinus thrombosis constituted an extension of thrombi in cavernous and lateral sinuses and the jugular vein.

TABLE VII  
DATA ON CASES OF SINUS THROMBOSIS IN TERMS OF TEETH EXTRACTED AND OF ASSOCIATED PATHOLOGIC CHANGES

CASE NUMBER	TEETH EXTRACTED*		DAYS SURVIVAL AFTER EXTRACTION	DAYS THROMBOSIS AFTER EXTRACTION	INTRAORBITAL ABSCESS	ADVANCING SUPPURATION AND/OR OSTEOMYELITIS	SINUSES THROMBOSED†					COMPLICATING INTRACRANIAL LESIONS						ORGANISMS CULTURED																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																								
	UPPER	LOWER					CAVERNOUS	SUP. PETROSAL	INF. PETROSAL	LATERAL	SIGMOID	ANTECEDENT MENINGITIS	TERMINAL MENINGITIS	CEREBELLAR ABSCESS	CEREBRAL ABSCESS	SUBARACHNOID HEMORRHAGE	SUBDURAL HEMORRHAGE																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																									
12			22	16	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

\*The sites of the extracted teeth indicated are the sources of origin of the spread of the infection. All the teeth removed in each case are listed in Table I.

†The sites of thrombosis are bilateral unless otherwise specified.



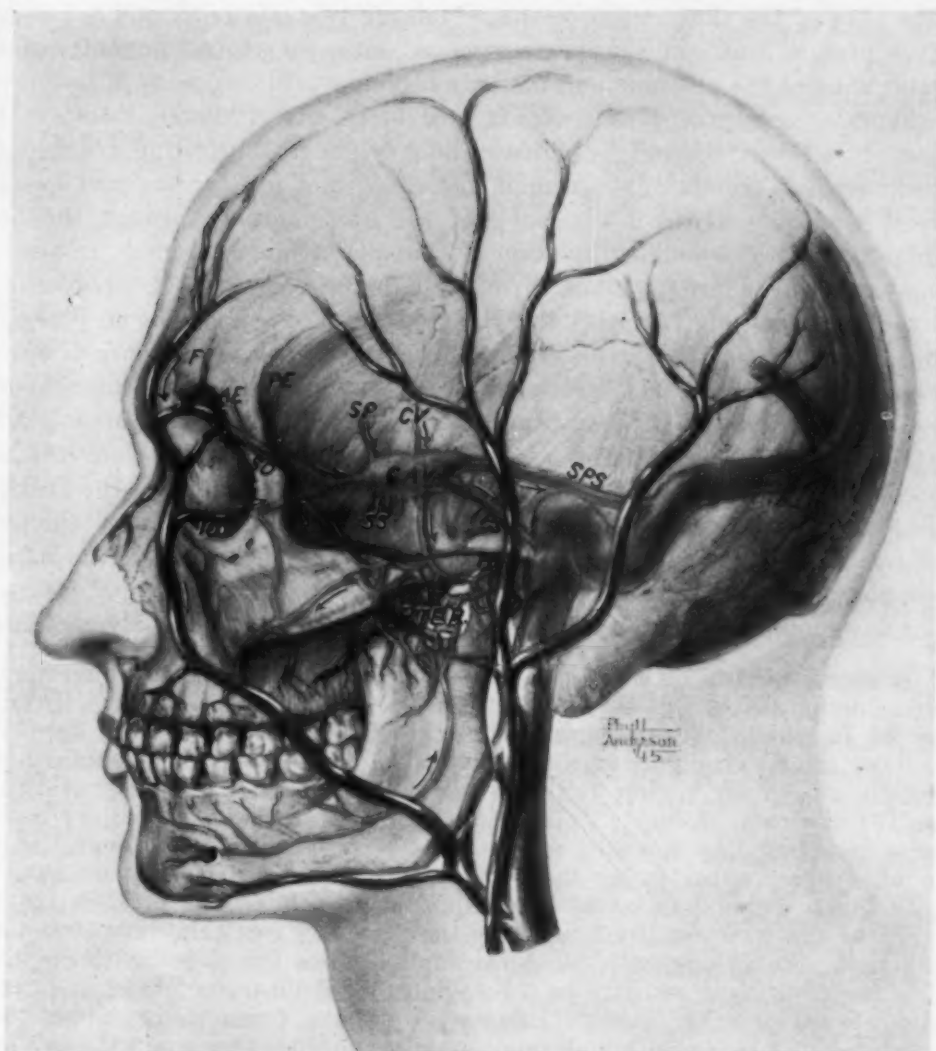


Fig. 32.—The venous tributaries of the cavernous sinus, including those from the teeth. The arrows indicate the direction of blood flow. Veins of the lower jaw drain via the inferior dental vein into the pterygoid plexus of veins (*PTER*), while those from the upper jaw drain in two directions: the more anterior ones into the anterior facial vein, and the more posterior ones into the pterygoid plexus.

The pterygoid plexus lies in the subtemporal and pterygoid fossae; it surrounds the lateral pterygoid muscle and covers the lateral surface of the medial pterygoid. In addition to the teeth mentioned, the pterygoid plexus drains the fauces, the soft palate, and the pharynx. In ascending infections complicating tooth extraction, the pterygoid plexus, together with the adjacent pharyngeal plexus (*PH*), is subject to thrombophlebitis. The infection usually reaches the cavernous sinus (*CAV.*) by way of the vein of Vesalius (present but not labeled) and often traverses the foramen of the same name, which is situated anteromedial to the foramen ovale; other routes followed from the pterygoid plexus are by way of the foramina ovale and lacerum. On the other hand, a thrombophlebitis of the pterygoid plexus may extend through veins which communicate with the inferior ophthalmic vein (*IO*).

In cavernous sinus thrombosis following infections of anterior teeth the thrombophlebitis tends to take the anterior route; i.e., via the anterior facial and its continuation, the angular vein (formed by the union of the supraorbital and frontal veins), and then through the orbit by way of the ophthalmic veins, especially the superior.

Tributaries draining into the cavernous sinus by way of the superior ophthalmic vein (*SO*) are from anterior facial structures (external nose, lips, forehead, eyelids, and cheek) and from the mucosae of the frontal sinus (*F*), the anterior ethmoidal cells (*AE*), the posterior ethmoidal cells (*PE*), and the upper part of the lateral nasal wall. The cavernous sinus also receives venous blood from the mucosa of the sphenoidal sinus (*SS*), from the superficial inferior cerebral veins (*CV*), and from the sphenoparietal venous sinus (*SP*). The sphenoparietal venous sinus drains diploe of the lesser wing of the sphenoid and veins of the dura mater. Blood leaves the cavernous sinus chiefly through the superior and inferior petrosal sinuses (*SPS* and *IPS*, respectively). (Partly after Turner and Reynolds.) AMM Neg. 84314.

at the base of the skull, while in the remaining two (Cases 22 and 23) the infective process was sufficiently invasive to cause severe osteomyelitis of the greater wing of the sphenoid and the sella turcica.

From a survey of these cases it is evident that in seven instances the venous blood was the only pathway along which the infection reached the cavernous sinus from an extracranial focus, and that in the remaining two the process was one of direct extension of the inflammation through the bony cranial wall with secondary invasion of the venous blood stream. In most of these cases the cavernous sinus thrombosis doubtless was the consequence of upward extension of phlebitis or of infected thrombi originating in pterygoid and pharyngeal venous plexuses (Fig. 32). This is the usual route of spread to the cavernous sinus from extracranial suppurative foci complicating dental infections: in a review of 74 instances of cavernous sinus thrombosis following dental infection, some initiated by tooth extraction, Childs and Courville concluded that the pterygoid route was the one followed in 32, and the facial in 9; in 3 there was a combination of the two, while in the remaining 30 the pathway traveled by the infection was not known. On occasion, cavernous sinus thrombosis may be due to spread of the infection from the pterygoid plexus to the inferior ophthalmic vein, and thence to the cavernous sinus (Barthélémy).

It seems worthy of emphasis that cavernous sinus thrombosis frequently occurs in the absence of severe extracranial suppurative cellulitis. Thus, in Case 24, in which all sinuses were thrombosed and in which death ensued in five days after extraction of a lower third molar, the only preliminary suppuration was in the region of the tonsil. Similar circumstances prevailed in Case 12: the only grossly visible suppuration was in the submaxillary region. Another, reported by Sproule, fits into the same category: following extraction of a lower third molar there was transitory drainage of pus from the empty tooth socket but no further suppuration; chills and fever set in five days later and were remittent until the time of death forty-six days after tooth extraction. At autopsy all the dural sinuses were found to be thrombosed. Aside from purulent exudate in the region of the pituitary gland and oculomotor nerve, no other abnormalities were noted. Cases of cavernous sinus thrombosis in which preliminary suppurative cellulitis was minimal have been reported also by Taylor and by Simpson. In Taylor's case a lower second molar was extracted for reasons not stated, while in that of Simpson there was local abscess due to an infected molar; in both instances the course lasted about one month.

Of importance in the clinical diagnosis of thrombosis of the pterygoid plexus is the presence of edema of the pharynx (Wilson). Pharyngeal edema was observed in three instances (Cases 12, 19, and 24), and if present in other cases was not recorded or was obscured by suppurative cellulitis in the vicinity. Abscesses of the throat and of the floor of the mouth appear to have been more frequent in this group than in cases without sinus thrombosis.

Evidence that the route of spread to the cavernous sinus may have been through anterior facial, and thence orbital veins, is to be found in only one instance (Case 22), but even in this case there is no proof that this was the sole route, or even the essential route, of thrombotic spread.

In the case in point an upper right first molar had been removed. Three days later the right eyelids and the right side of the nose became edematous. This was regarded as evidence of thrombosis of the angular vein. Cellulitis

soon spread to the opposite side of the face and into tissues of the mouth, requiring incision and drainage of palpebral and oral abscesses. Paranasal sinuses also were purulent. Thirty-five days after extraction the conjunctivae of the right eye became edematous, and the eyeball unduly prominent. At autopsy on the forty-eighth day after extraction the orbit was free from involvement but the cavernous and other dural sinuses were thrombosed. Abscess of subtemporal fossa and osteomyelitis of the greater wing of the sphenoid were also disclosed post mortem.

In another instance (Case 18), one of cavernous sinus thrombosis with fatal outcome in twenty-one days after extraction of the lower left first, second, and third molars (L-14, 15, 16), an ophthalmic vein (whether superior or inferior was not specified) was found at autopsy to be thrombosed. Proptosis had occurred on the seventeenth day. The thrombosis of the ophthalmic vein may have represented a forward extension from the cavernous sinus or it may have been the result of direct spread from the pterygoid venous plexus, with subsequent cavernous sinus thrombosis.

An authentic case of cavernous sinus thrombosis following extraction of an upper left central incisor (L-1) has been reported by Koepf, Rosedale, and Learn. The infection was borne to the cavernous sinus by way of orbital veins. An independent suppurative process extended backward from the region of the extracted tooth to the subtemporal fossa, encompassing the pterygoid venous plexus but not leading to thrombosis of this structure. Strictly speaking, this case does not belong with the series reported in this paper since advancing alveolar infection was present at the time of tooth extraction.

The patient, white, aged 62, developed an alveolar abscess in the region of the upper left central incisor (L-1) and three days later extracted the tooth himself. On the eighth day after extraction there was bilateral proptosis. Death occurred nine days later. At autopsy it was apparent that the infective process had followed two routes: (1) through ophthalmic veins (which were thrombosed bilaterally) to the cavernous sinus, and (2) through soft tissues of left cheek (where an abscess 1.5 cm. in diameter was located) into the subtemporal fossa (where there was an abscess 4.0 cm. in diameter), encroaching on the posterior wall of the pharynx and extending as far back as the petrous part of the temporal bone. In addition, the appearances indicated that the purulent exudate had extended along the sheath of the mandibular division of the trigeminal to the gasserian ganglion and thence subdurally, where a plaque of fibrinopurulent exudate, about 4.0 cm. in diameter, was located. The internal carotid in the region of the cavernous sinus was thrombosed. Paranasal sinuses, leptomeninges, and brain were unaffected.

A case of cavernous sinus thrombosis following extraction of upper incisors (L-1, 2), in which the infection extended posteriorly through orbital veins, has been described also by Morgenstern.

It is generally stated that cavernous sinus thrombosis from anterior facial sources tends to occur more rapidly than does that from an infected pterygoid venous plexus. There are, however, exceptions to the rule. Although the average time interval between extraction and cavernous sinus thrombosis in this series of cases was sixteen days (and total course twenty-four days), the thrombosis occurred in five days after extraction in Cases 20 and 24, and in eight days in Case 19. In the case of Koepf, Rosedale, and Learn, referred to in the preceding paragraph, in which the infection spread to the cavernous sinus via



ophthalmic veins, the time which elapsed between extraction and the development of cavernous sinus thrombosis was eight days.

*Lateral (or transverse) sinus thrombosis* occurred in three instances (Table VII). In two (Cases 25 and 26) the route pursued by the infection was through the skull; in the other (Case 27) the pathway is not evident but is considered to be by way of the general circulation. The background of the infections is much the same as that described on previous pages, the only significant difference being that the infections extended more posteriorly than usual, inducing osteomyelitis of the petrous bone in one instance (Case 25) and suppurative cellulitis of the postauricular scalp in another (Case 26). Cerebellar abscess complicated lateral sinus thrombosis in one of these (Case 26).

It is of interest that mastoiditis after tooth extraction may also lead to temporal lobe abscess and, while sparing the lateral sinus, may give rise to thrombosis of the superior petrosal and cavernous sinuses. Such a case has been reported by Bolton.

In the case in point, the patient, aged 48, had had an upper left molar extracted because of caries. After an unstated interval of time an alveolar abscess was incised. The tissues behind the left sternomastoid muscle soon became much indurated. A convulsive seizure, predominantly of the right side, was the first evidence of intracranial involvement. Subsequently an otitis media became evident on the left side, and mastoidectomy was performed. The duration of the illness is not known. At autopsy there was epidural and subdural pus in the region of the left lateral sinus, a left temporal lobe abscess and purulent thrombosis of the superior petrosal and cavernous sinuses. In addition, a large subtemporal abscess was present on the left side. It was concluded that the infective process reached the mastoid by way of emissary veins and that the temporal lobe abscess and the sinus thrombosis were complications of the mastoiditis.

#### SUMMARY AND CONCLUSIONS

1. Twenty-seven cases of fatal intracranial complication of tooth extraction, and one of transverse myelitis, are presented in some detail since relatively few such cases are on record. The extractions are believed to have initiated or precipitated the infective process in virtually all of the cases.

2. The teeth were removed because of periapical abscess, caries, impaction, malposition, painful eruption, and for other reasons. Cases in which teeth were extracted in order to drain an already advancing osteomyelitis of the jaw were not included in this series.

3. The mouth was in poor hygienic condition in only 8 of the 28 cases.

4. In 19 of the cases only one tooth was extracted, an observation which indicates that, in this series at least, the danger of fatal intracranial complication lies elsewhere than in multiple extraction. In cases in which the greatest number of teeth had been extracted there was no evidence that bacteremia ensued.

5. Upper teeth were removed in exactly the same number of cases as lower teeth. Direct spread of the infective process to the intracranial cavity was encountered more often after extractions from the upper than from the lower jaw (10 as against 6), while in hematogenous infections the reverse was the case (9 to 5). A review of the literature reveals that fatal complications of



dental infection of the lower jaw are approximately twice as frequent as those of the upper jaw, and that fatal infections of the left side outweigh those of the right side by 3 to 2.

6. Of teeth extracted, the molars predominated. Only molars had been extracted in cases in which cavernous sinus thrombosis ensued (except for one in which a bicuspid was also removed). The tendency for infection in the vicinity of molar teeth to lead to intracranial complications doubtless is to be ascribed to anatomic relations: not having free access to the oral cavity or to the exterior as in the anterior part of the jaw, the pus tends to collect between the muscles of mastication and to spread rapidly upward in fascial planes.

7. In some of the cases the collection of pus at the base of the skull was so deeply situated that its presence was not recognized or was not reached by surgical means.

8. From a bacteriologic standpoint the most frequently encountered organism in hematogenous infections was the streptococcus, and in the infections reaching the intracranial cavity by direct spread, the staphylococcus. Bacteremia is judged to have occurred as the immediate result of extraction in 7 cases, shortly after extraction in association with fulminant cellulitis of the jaw in 2, approximately one month after extraction as the result of surgical intervention in 2, and at undetermined times in 3.

9. Spread of the infective organism to the intracranial cavity by way of the general circulation occurred in 10 instances, and to the spinal cord in one. In 7 of the 11 there was brain abscess, and in 4, leptomeningitis, choroiditis, lateral sinus thrombosis, and transverse myelitis, respectively.

10. Direct spread of the infective process to the intracranial cavity occurred in 18 cases. In 8 of these there was suppurative cellulitis which spread to the base of the skull, producing osteomyelitis of the greater wing of the sphenoid bone. Brain abscess due to direct spread of the inflammation through the bony cranial wall occurred in 7 instances. In 7 of the 9 cases of cavernous sinus thrombosis the venous blood stream was the only pathway along which the infection reached the sinus from the extracranial focus, while in the remaining 2 the process was one of direct extension of the inflammation through the bony cranial wall with secondary invasion of the venous blood stream. Intraorbital abscess occurred in 6 instances. Of paranasal sinuses involved, the sphenoidal came first and the maxillary second.

Captain Henry Goldman, D. C., rendered invaluable assistance in considering the individual cases from the dental standpoint, and Major Herman Semenov, M. C., was of great help in locating the positions of the paranasal sinuses in Fig. 30.

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## CASE REPORTS FROM THE ARMY INSTITUTE OF PATHOLOGY

CAPTAIN HENRY M. GOLDMAN, D.C., AND MAJOR HUGH G. GRADY, M.C.

### Case 1

#### Spindle-Cell Carcinoma of the Lip

A 31-year-old white soldier sustained a cigar burn of the lower lip while engaged in a friendly scuffle. The burn did not heal well and two months later he was hospitalized. At that time there was a large ulcer, approximately 1.5 by 1.0 cm. on the vermilion border of the lower lip at the site of the burn. The base of the ulcer was dirty gray and had little tendency to bleed. Beneath the ulcer was a firm mass extending into the soft tissues of the lip for about 8 mm. The patient stated that he had not observed any tumor prior to the burn. Several dark-field examinations revealed no spirochetes nor was there serologic evidence of syphilis. The regional lymph nodes were not palpably enlarged. A V-shaped excision of the tumor was performed. The growth was composed of firm, homogeneous, gray-white tissue, and measured 1.4 by 1.1 by 0.8 cm. Numerous sagittal sections did not indicate any further extension into the substance of the lip. (Fig. 1.)

The floor of the ulcer consisted of a thin layer of inflammatory cells covering a moderately cellular neoplasm which superficially resembled a sarcoma. The tumor cells were polymorphous or spindle-shaped, in some places arranged in interlacing bundles and surrounded by large amounts of collagen. The nuclei were large and oval or elongated with prominent acidophilic nucleoli. Mitotic activity was moderate. No evidence of keratinization was noted nor were any intercellular fibrils seen. There was moderate hyperplasia of the covering epidermis but no indication of neoplastic change. Although the tumor had superficially invaded the muscle of the lip, excision was thought to be complete. The diagnosis at this time was spindle-cell epidermoid carcinoma. (Fig. 2.)

The patient remained well for seven months; then he noted pain in the right lower molar area. In a few days a hard, painless swelling, 1.5 cm. in diameter, appeared in the right submandibular region. The mass was incised and a large quantity of pus evacuated. A second biopsy was examined and a diagnosis of secondary anaplastic squamous carcinoma was made. This tumor was characterized by sheets and masses of large, round, polygonal or elongated cells with large, usually vesicular nuclei and prominent nucleoli. Spindle cells were not a feature as in the primary growth. Keratinization was not observed, but the squamous nature of the cells was apparent (Fig. 3). One week following this diagnosis a segment of the right mandible including the second bicuspid, the first and second molars, as well as the tumor was

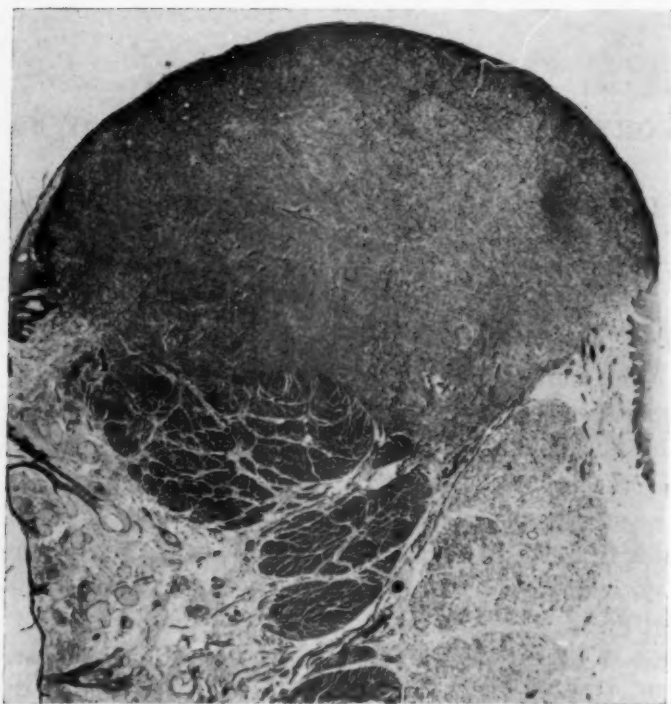


Fig. 1.—Sagittal section of primary tumor showing superficial invasion of muscle. (Neg. 84599,  $\times 10$ .)

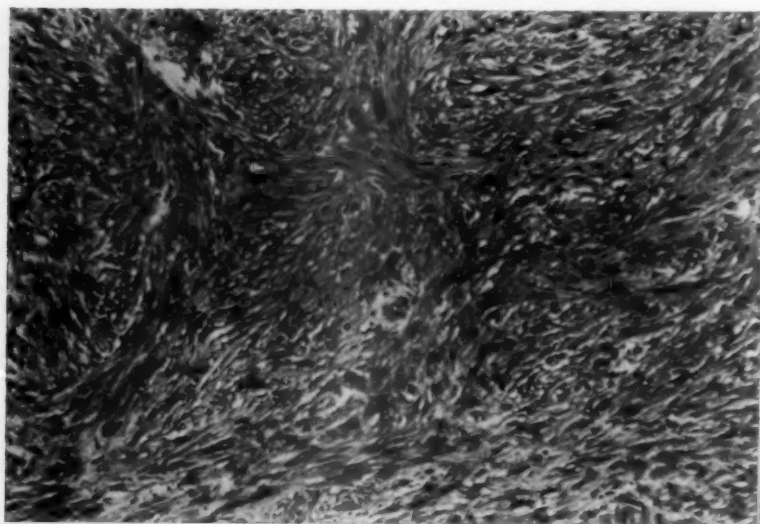


Fig. 2.—The primary tumor is composed of interlacing bundles of spindle cells and resembles a sarcoma. (Neg. 82220,  $\times 175$ .)



resected. The entire specimen measured 7 by 6 by 6 cm. The tumor was partially necrotic but had invaded the submaxillary and sublingual glands and the mandible. There is doubt that the tumor was completely removed. Fifteen months after the original diagnosis was made the patient is presumed to be alive.

This case presents a number of features not ordinarily encountered in carcinoma of the lip. The patient was only 31 years old, considerably under the age at which this type of tumor usually is seen. Spindle-cell epidermoid carcinoma of the lip has been reported previously, notably by Martin and Stewart,<sup>1</sup> but is a distinctly uncommon histologic entity. The resemblance to sarcoma in the primary growth was sufficiently striking to give rise to some difference of opinion among several pathologists, but the true character is obvious in the metastasis. The relation of thermal trauma to the onset of tumor seems reasonably established in point of time and the patient was insistent that no tumor had been noted prior to the burn. Any causal relationship, however, between trauma and tumor is purely speculative. It is worthy of note that in a number of Martin and Stewart's cases there was history of previous trauma, chiefly from irradiation. There seems little doubt, in view of the rapid appearance of the primary lesion and the almost equally rapid recurrence, that this is a highly malignant tumor.

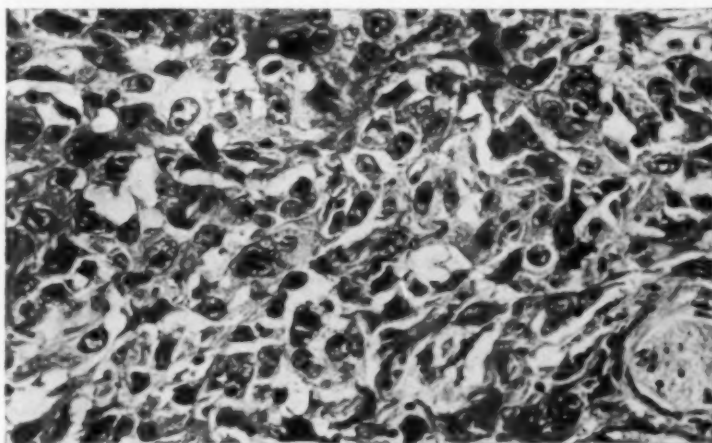


Fig. 3.—The recurrent tumor is composed of large irregular cells with little collagen. The squamous character is apparent. (Neg. 84651,  $\times 400$ .)

## Case 2

### **Adenocarcinoma of Gingiva Infiltrating the Mandible and Metastasizing to a Regional Lymph Node**

A white man, 33 years old, noticed a small asymptomatic swelling of the gingiva following the extraction of a left mandibular premolar. The swelling gradually increased in size until, in three months, it became as large as a "hickory nut." The entire mass was removed for biopsy.

Microscopic examination of the tissue disclosed ulceration at one point in the oral mucosa with the tumor composing the base of the ulcer. The lesion

proper consisted of small epithelial cells with round, moderately hyperchromatic nuclei and relatively small amounts of cytoplasm. These cells were arranged in acini of various size and shape, separated by broad bands of connective tissue (Fig. 4). Many acini contained red blood cells while others were

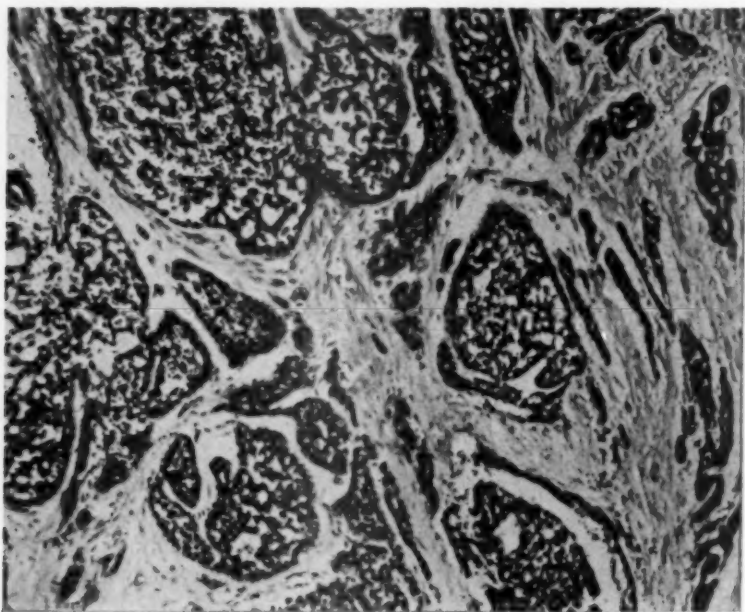


Fig. 4.—Adenocarcinoma; note the acinar arrangement of tumor cells. (Neg. 81614,  $\times 145$ .)

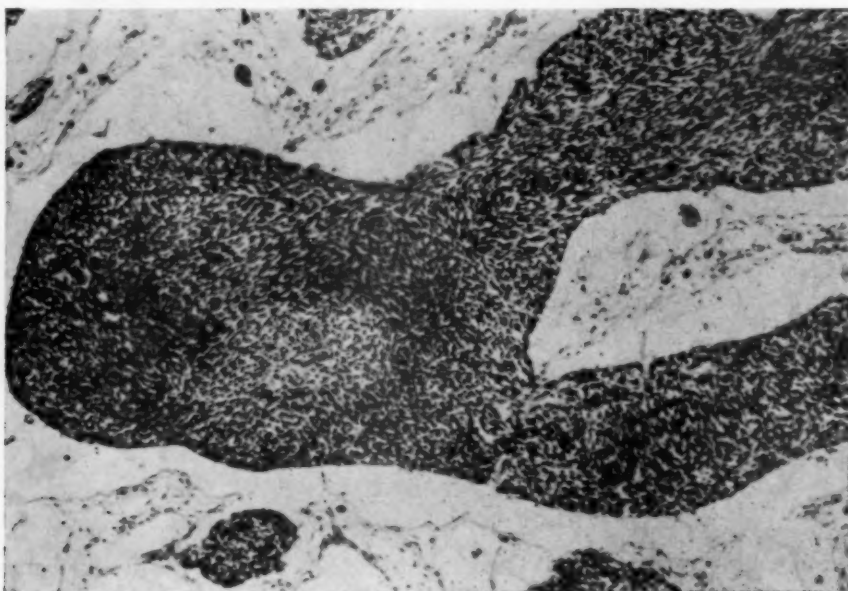


Fig. 5.—Section showing cells closely packed. (Neg. 81610,  $\times 145$ .)

filled with a blue-gray mucinous material. Some cells were spindle-shaped and closely packed, with no acinar arrangement (Fig. 5); others at the periphery were cuboidal or low columnar. The stroma consisted of loose, edem-

atous connective tissue. The tumor infiltrated the surrounding tissue, and in the margin of the lesion, one thin-walled, rather large capillary contained tumor cells.



Fig. 6A.—Swelling of face caused by tumor. (Neg. 81459.)



Fig. 6B.—Intraoral appearance of tumor. (Neg. 81462.)

A diagnosis of adenocarcinoma was made.

Four months later the tumor recurred and assumed much larger proportions extending in the soft tissue across to the opposite side (Figs. 6A and 6B).

The entire left body of the mandible was involved; the radiograph showed a diffuse osteolytic process with destruction of the alveolar border (Fig. 7). The involved portion of the mandible was resected together with the surrounding tumor.

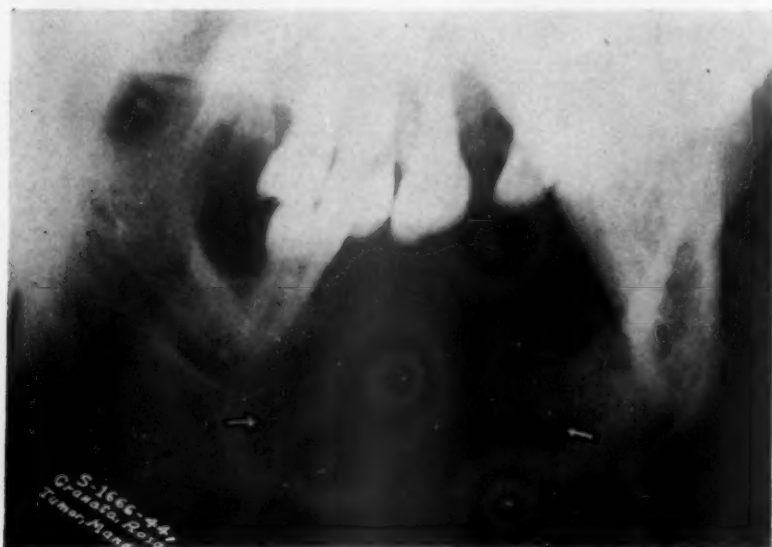


Fig. 7.—X-ray showing osteolytic defect caused by tumor infiltration. (Neg. 81461.)



Fig. 8.—Gross specimen; the lobulated appearance of the tumor is evident. (Neg. 81457.)

The gross specimen (Fig. 8) was composed of the body, the angle, and about 3 cm. of the ramus. Only the third molar, which was rather loose, was present. Embracing the entire resected portion of the mandible was a solid tumor which was firm, pinkish-red, and somewhat lobulated. On the lateral aspect of the angle of the mandible and beneath it were discrete glands. The tumor infiltrated the mandible, only a small portion of the inferior border



remaining intact. In the bone the tumor was composed of lobulated, grayish-yellow, soft, homogeneous material. In one area the muscles attached to the mandible were invaded.

The tumor had essentially the same microscopic features as the original growth. Acinar or compactly arranged, small, round, moderately hyperchromatic cells in a lobular arrangement were characteristic of the tumor. The mandible was invaded as well as the muscles attached to the bone. The tissue of one lymph node was entirely replaced by tumor (Fig. 9).

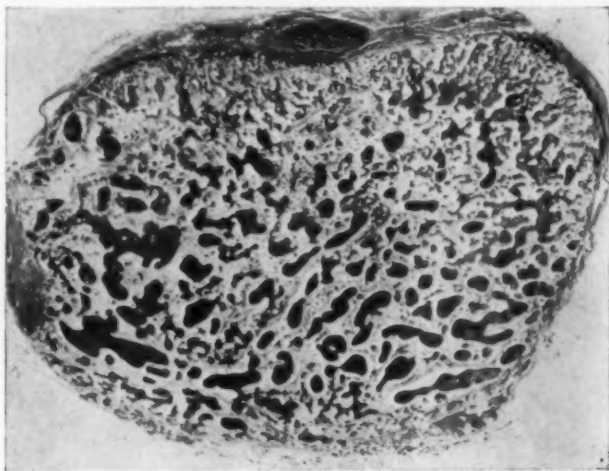


Fig. 9.—Tumor tissue in lymph node. (Neg. 81751,  $\times 12$ .)

Some question has existed as to whether a tumor of this nature should be regarded as an entity. It has been thought to be a mixed tumor, an adenocarcinoma derived from the salivary and mucous glands, an adenoid cystic carcinoma resembling adenoid cystic epithelioma of the skin, and adenoamantinoma. It may appear in the oral cavity, the nose, the oropharynx, or the nasopharynx. Some investigators believe that metastasis to distant organs from this tumor is usually the cause of fatality, while others regard local recurrence or invasion of contiguous structures as more often the cause of death. All agree that it is a slow-growing tumor which infiltrates and tends to recur.

Stewart, in a report by Watson,<sup>4</sup> states that for the most part these tumors appear to be derivatives of the minor salivary glands or of surface epithelium and ducts differentiating in a glandular direction. He finds that few have a tendency to differentiate in the direction of so-called mixed tumors; the majority have a structure like that of adenoid basal-cell tumors of the skin. He believes they should be termed adenocarcinomas of salivary gland origin.

It is difficult to estimate the malignancy of this tumor from its morphology. It resembles basal-cell carcinoma, the predominant cell being small and round, hyperchromatic, with little cytoplasm. Pronounced atypism of the epithelium is not characteristic of the tumor nor has it a great number of mitotic figures; however, definite invasiveness is usual and metastasis to the regional lymph nodes occurs, although not commonly, to judge from reports in the literature. In a case reported by Sharnoff and Lisa<sup>3</sup> the patient died from metastases to

the regional lymph nodes and the lungs. Watson<sup>4</sup> stated that in a series of twenty-two cases followed for more than five years, 45.4 per cent of the patients were dead, 9.1 per cent alive with the disease, 36.4 per cent alive and well, and the remaining 9.1 per cent unaccounted for. This series of cases is the largest reported and, therefore, the most informative. Thus it would seem that, although the morphologic appearance of this tumor suggests benignity, it is best to regard it as a malignant growth which should be given prompt and radical treatment.

Of interest in the case presented is the infiltrative growth of the lesion and its rapid recurrence after the initial operation. The tumor infiltrated the mandible and metastasized to a regional lymph node. This type of tumor should be regarded as a well-defined entity to be distinguished from the malignant mixed tumor and also from the alveolar carcinoma often seen in the sub-maxillary gland.

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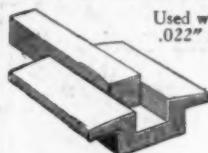
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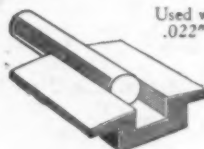
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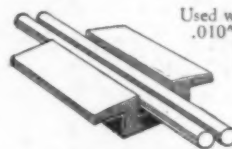
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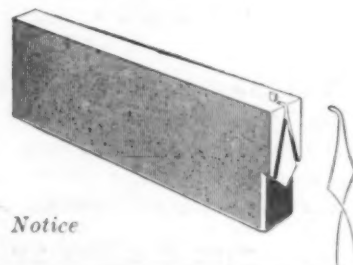
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APRIL, 1945

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# Massachusetts General Hospital Number

THE CLINIC OF THE MASSACHUSETTS GENERAL HOSPITAL AND  
THE HARVARD SCHOOL OF DENTAL MEDICINE

## Volume IV

KURT H. THOMA, D.M.D.,\* HENRY D. HOWE, D.M.D.,† AND  
MARTIN WENIG, D.D.S.‡

WITH CONTRIBUTIONS BY  
DAVID WEISBERGER, D.M.D., M.D.,‡ AND IRA D. NATHANSON, M.D.§

## INTRODUCTION

We are presenting herewith the fourth Massachusetts General Hospital Number with cases treated in the second half of 1944. We are also publishing a preliminary report on the obliteration of bone wounds by means of fibrin foam. Fibrin foam has been developed by the Department of Physical Chemistry, Harvard Medical School, for arresting operative hemorrhage. It has been used by us to fill bone cavities resulting from the removal of unerupted teeth or excision of moderate-sized cysts. The foam, soaked in a mixture of thrombin and tyrothricin, was loosely placed into the wound which was then closed with sutures. This method was developed to shorten the postoperative care.

For the fixation of fractures of the mandible a peripheral bone clamp has been developed to replace the pins inserted into the bone in skeletal fixation. This method of internal clamp fixation is particularly useful for the edentulous posterior fragment in fractures at the angle of the jaw. Instead of inserting two pins into the ramus, a clamp is attached here where the bone is thin and unsatisfactory for pin fixation. The remainder of the appliance is attached to the pin inserted through the skin to fasten the clamp, and two pins placed into the anterior fragment in the ordinary manner.

The large number of condylar fractures treated during the last two years have given us an opportunity to analyze these cases, and list the complications found. The results of this study are presented in tabular form. They include four old fractures with malunion complicated by pseudarthrosis in one, and ankylosis of the jaw in three; this supports our contention that the conservative treatment in vogue does not give the consistently good results proclaimed by its advocates. A method, therefore, was developed by us for the reduction and fixation by interosseous wiring of badly displaced condylar fractures, and interosseous wiring combined with skeletal fixation for fracture dislocations.

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The technique of these methods has been described in a recent article in the *Journal of Oral Surgery*,\* but a case in which both these methods were used will be presented here.

Experiments with penicillin therapy in neck infections of odontogenic origin have been carried out during the period covered by this report; the results, however, will not be published until the series is large enough to allow us to make valuable deductions.

The remaining part of the volume contains a variety of cases we thought would be of interest to our readers.

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## A NEW METHOD OF SPACE OBLITERATION AFTER ODONTECTOMY

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### A PRELIMINARY REPORT

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KURT H. THOMA, D.M.D.

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The postoperative care following odontectomy, especially after the removal of retained mandibular third molars, has always presented a problem to the oral surgeon. The many procedures that have been advocated in the past, and the diversity of recommendations made by those who have written on the subject more recently, is testimony for the fact that no uniformly satisfactory method of treatment has yet been found.

Many operators believe in allowing the wound to heal by granulation. Some permit a blood clot to form and apply gauze between the teeth to protect it until bleeding stops, hoping that part of the clot will remain and organize to shorten the healing time, while food and secretions are removed by daily or less frequent mildly antiseptic irrigations. Others believe in inserting a dressing into the wound; generally this dressing is medicated either with an antiseptic or with drugs that relieve pain. These dressings must be changed at regular intervals. In both instances the aftercare requires ten to twenty days.

To shorten this period of convalescence, which in many instances is a great inconvenience to the patients and the cause of a certain amount of suffering and economic loss, many dental surgeons have tried to promote more rapid healing by closing the wound with sutures. This procedure has been satisfactory only in a small percentage of cases; in many instances it has promoted swelling and ecchymosis of the skin due to subcutaneous hemorrhage. Other times, suturing the wound encloses infection that, later, after the patient has been discharged, may cause bone necrosis and submaxillary and submandibular abscesses.

The development of a method, therefore, which allows the odontectomy wound to be closed with safety is one of the important needs in oral surgery. Three factors seem to be involved:

1. Because absolute asepsis is difficult to obtain and maintain during the operation, a bactericidal and bacteriostatic agent is needed.

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\*Thoma, K. H.: The Treatment of Fractures and Fracture Dislocations of the Mandibular Condyle, With Description of a Method for Open Reduction and Internal Wiring of These Fractures, and Another for Skeletal Fixation of Dislocations, *J. Oral Surg.* 3: 3, 1945.



2. Because secondary hemorrhage, although generally only slight, is common during the first twenty-four hours and sometimes occurs after the hemostatic effect of the local anesthesia subsides, a local hemostatic agent preventing disfiguring ecchymosis is a desirable addition.

3. To prevent the breakdown of too large a blood clot, thus furnishing an excellent medium for bacterial growth, an agent to eliminate space must be found.

The bactericidal problem seemed near solution when the sulfonamides came into use. I am sure that both the prophylactic and therapeutic use of local implantation of either sulfanilamide or sulfathiazole decreases the frequent aftereffects of odontectomy, namely, pain, swelling, and infection. According to Millhon, Austin, Stafne, and Gardner,\* when the drugs are applied on a dressing, the healing time is not greatly expedited. If they are placed into the wound, and the wound closed by suture, as recommended by Moorehead,† resorption takes place too rapidly. Also, the local use of sulfanilamide powder in a wound is said to increase bleeding; when the wound is closed, this will produce ecchymosis, which, as already stated, is one of the objections to be eliminated. If sulfonamides are used, the choice of the particular type is important. Since sulfanilamide has little effect on staphylococci and beta hemolytic streptococci, sulfathiazole should be used if the infection is due to the latter organisms. Cones‡ containing both sulfanilamide and sulfathiazole have the added advantage of dissolving more slowly than sulfanilamide alone. Since the blood clot does not break down until the second or third day, local sulfonamide therapy of any type is not very efficacious, as the drugs resorb long before the need for a bactericidal agent is the greatest. Griess§ found that the local effect lasts about thirty-six hours.

To arrest bleeding and prevent secondary hemorrhage as well, experiments with tablets containing thrombin and tyrothricin|| have been made. These tablets have been used in several cases in which the patient had either a history of excessive bleeding after tooth extraction, or in cases in which excessive bleeding was noticed after removal of the tooth, as in cases of local inflammation due to infection. The findings in sixteen cases are tabulated below.

*Mr. C. C. R.*—May 11, 1944. This patient had had trouble previously with bleeding after extractions. Six incisors were extracted, and alveoloplasty done. Tablets were put into all extraction sockets. Patient had no hemorrhage or complications.

*Mrs. A. B.*—May 15, 1944. Right and left first, second, and third molars extracted. Because of gingivitis, thrombin and tyrothricin tablets were used. Healing was uneventful.

*Mrs. A. G.*—July 14, 1944. Right upper first molar had a pulp infection; it was extracted. There was considerable hemorrhage from the socket, which was arrested with two tablets. Healing was uneventful.

\*Millhon, J. A., Austin, L. T., Stafne, E. C., and Gardner, B. S.: *J. Am. Dent. A.* 30: 1839, 1943.

†Moorehead, F. B.: *J. Am. Dent. A.* 29: 2162, 1942.

‡Supplied through the courtesy of the Novocol Manufacturing Company, for clinical experimentation.

§Griess, F.: *J. Dent. Research* 21: 3, 1942.

||These tablets were supplied through the courtesy of Sharp & Dohme.

*Mrs. F. J. T.*—July 17, 1944. This patient had had trouble with bleeding before. Right upper central incisor was extracted; one tablet was used. No postoperative complications.

*Mr. D. McC.*—July 27, 1944. Right upper root of second molar extracted. There was arterial bleeding. Two tablets were applied and bleeding stopped immediately. Healing was uneventful.

*Mr. G. P.*—Aug. 14, 1944. Left lower first and second molars were extracted. Tablets were used to stop excessive bone hemorrhage after the extraction.

*Dr. H. G.*—Aug. 30, 1944. Right lower second molar was extracted. Due to pyorrhea there was considerable bleeding which was arrested by the use of one tablet, placed between the bone and gingiva.

*Mr. A. D.*—Sept. 7, 1944. Patient had Hodgkin's disease and hemorrhagic tendencies. There was an ulceration of the gingiva at the left lower second molar. This tooth was removed under gas-oxygen-ether anesthesia. Eight tablets were inserted into the socket and held there by iodoform gauze. Healing was uneventful.

*Mr. R. B.*—There was a history of extensive hemorrhage from tooth extraction. The patient had slight gingivitis and infection of the six upper anterior teeth. These teeth were extracted and alveoloplasty done. Two tablets were put into each socket (12 tablets in all), and the mucoperiosteum replaced, closing the incision with a continuous suture. Recovery was uneventful.

*Mr. D. D.*—Sept. 13, 1944. Patient had had pains on the left side of the face and neuralgia over the left side of the head, with a tired feeling. X-rays showed advanced pyorrhea. Left upper second molar had a periodontal abscess, and the left lower second premolar, a large cavity with pulp involvement. These two teeth were extracted. Tablets were inserted into the extraction wounds. Recovery was uneventful.

*Mrs. A. D.*—Sept. 21, 1944. Four extractions of teeth with extensive pyorrhea pockets. Tablets were inserted after extraction. Recovery was uneventful.

*Mr. H. H.*—Sept. 25, 1944. There was a periodontal abscess between the roots of the right upper third molar. This tooth was extracted, and the socket débrided. One tablet was applied. Recovery was uneventful.

*Mrs. C. B.*—Sept. 27, 1944. Two impacted (class 3) teeth, right and left lower third molars, were excised. Three tablets were placed into each wound, and a pack placed between the jaws. The patient had secondary hemorrhage during the night on the right side, which her local dentist was unable to stop. I visited the patient and packed both wounds with gauze and Monsel's salt. Another hemorrhage occurred two days later. The healing on the left side was uneventful. The bleeding on the right was from a gingival vessel not affected by the tablets placed into the bone wound.

*Mr. W. G.*—Nov. 17, 1944. The patient had a chronic pericoronal infection with pain and bleeding for several days. The left lower third molar was excised, and two tablets inserted. A pack was placed between the jaws. Healing was uneventful.

*Miss A. P.*—Nov. 18, 1944. The right lower third molar was extracted. There was considerable hemorrhage. Two tablets were used, the bleeding stopped, and healing was uneventful.

*Mr. F. R.*—Jan. 18, 1945. This patient has had severe and prolonged bleeding after previous extractions, and was very apprehensive regarding his teeth. His physician advised removal of all remaining upper and lower teeth (18 in all). The clotting time was 5 seconds, and the bleeding time, 5 minutes, which is a high normal. The operation was performed in the hospital under gas-oxygen-ether anesthesia. There was excessive bleeding from the extraction wounds. An alveoloplasty was performed, and from one to three tablets were inserted into each bleeding socket (32 tablets in all). The gingiva was approximated and sutured with continuous suture (Fig 322). At the end of the operation the bleeding had stopped completely. The patient had no secondary hemorrhage.



Fig. 322.—Alveoloplasty after full mouth extraction. Incision closed by continuous suture after inserting thrombin and tyrothricin tablets into each socket.

In all these cases the bone wounds were relatively small, representing mainly the sockets in which the roots of the teeth were imbedded. In odontectomy when completely retained canines or third molars are excised, the bone wound is considerably larger, first, because some bone had to be removed to get access to the tooth, and second, because the cavity contained the entire tooth, crown as well as roots. A blood clot in so large a cavity will not survive; the prevention of a large clot therefore, seems to be the most important factor in achieving healing by first intention. Austin, et al., have pointed out that the greatest benefit of a dressing in a bone cavity created by the removal of an unerupted tooth is the displacement of blood from the central portion of the wound. If the size of the clot is reduced, they stated, the blood supply from the surrounding bone can more easily maintain vitality of the clot. Some years ago the use of bone wax was recommended for the obliteration

tion of space and to decrease the size of the clot in wounds to be closed by suture. I have found that bone wax is generally expelled as a foreign body.

In looking for a substance which would not cause any biologic reaction, I came across "fibrin foam," developed in the Department of Physical Chemistry at Harvard Medical School. Foam saturated with thrombin can be used to arrest hemorrhage. Being made from normal human plasma proteins, it can be left in the wound, and, therefore, seemed to have the qualities sought for in a substance to obliterate space and to decrease the size of the blood clot. The thrombin also seemed useful to arrest bleeding and to prevent secondary hemorrhage. All that was needed in addition was the incorporation of a bacteriostatic agent. In the experiments, tyrothricin was used (Fig. 323).

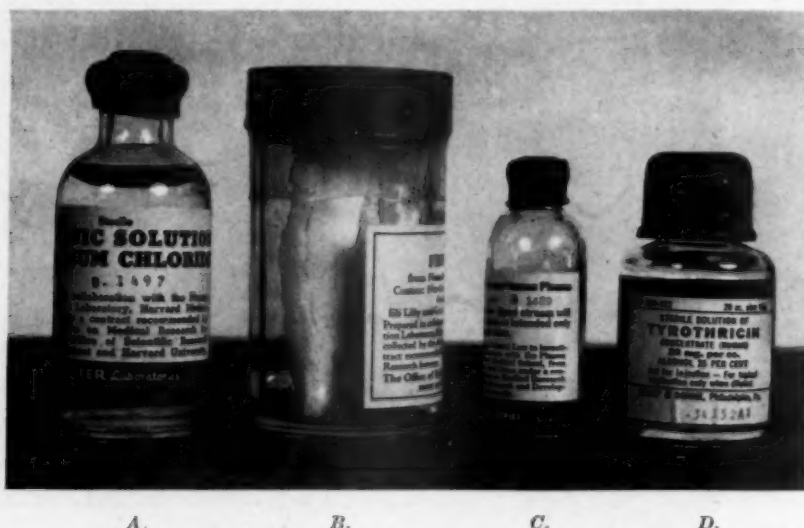


Fig. 323.—Fibrin foam and thrombin. B, Fibrin foam; C, thrombin from human plasma to be dissolved in A, isotonic solution of sodium chloride. To this is added the same amount of D, tyrothricin.

The following procedure was carried out:

*Selection of Cases.*—Only cases of mandibular third molars or maxillary canines that are completely unerupted, and which are not involved by infection, are considered suitable for space obliteration.

*Preparation for the Operation.*—In order to use fibrin foam successfully, the operation should be performed under aseptic conditions. Asepsis can be fairly well maintained under general anesthesia (gas and oxygen excepted), as well as under local anesthesia. In both instances atropine,  $\frac{1}{100}$  grain, should be administered to arrest salivation, or suction may be used to remove mouth secretions, but the suction tip should never be put into the wound to remove blood after having been used to remove mouth secretions. The mucosa should be properly prepared by spraying with hydrogen peroxide, diluted 1:2 with water, to destroy anaerobic bacteria. The patient should also rinse with, and hold in the mouth for three minutes, a solution of metaphen, 1:2,500, to reduce the number of pyogenic organisms. Just before the incision is made, a liberal quantity of tincture of zephiran, 1:1,000, is applied to the area to be operated on. After this, gauze may be placed lingual to the jaw. If saliva is



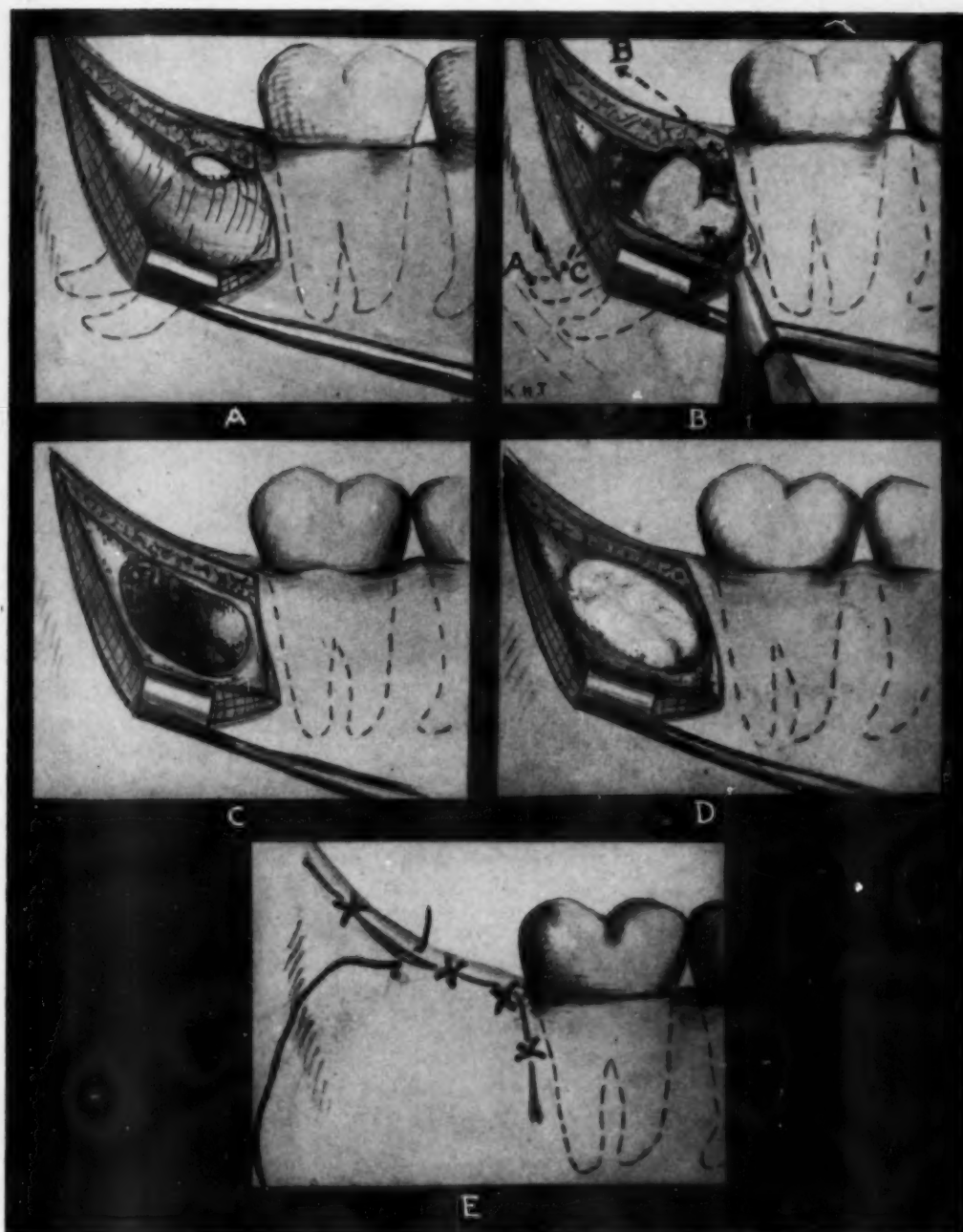


Fig. 324.—*A*, Retraction of the mucoperiosteal flap and exposure of the mandible.  
*B*, Bone removed to expose the crown of the tooth. A space is made at the distal surface of the impaction to facilitate its rotation in the direction of *B*. This takes care of the impacted mesial cusp, and the curvature of the apices.  
*C*, Bone cavity after removal of tooth.  
*D*, Fibrin foam inserted into the bone cavity.  
*E*, The wound closed by interrupted dermalon sutures.

still flowing, a small sponge is placed over the outlet of the parotid duct, and covered either by a retractor to hold out the cheek, or with the retracting finger. The patient is warned that he must not move, talk, close the mouth, or expectorate until the wound is closed.

*Operation.*—The operation is performed by any technique which allows protection of the wound so as to exclude contamination. The incision must be made so as to allow complete closure after the tooth has been removed. It must not be carried into the gingival crevice; tissue (gingival margin) must be left around the second molar (Fig. 324, *A*). Sufficient bone should be excised to make removal of the teeth easy (Fig. 324, *B*), or the tooth should be sectioned so that it can be extracted in parts without using undue force. After the removal of the tooth, the wound is débrided; remnants of the dental follicle are removed and the bone margins carefully inspected and rough edges smoothed (Fig. 324, *C*).

The bone cavity is next filled with fibrin foam.\* In the cases to be cited the foam was soaked in a solution of thrombin and tyrothricin,† mixed 1:1; the former in half the dilution ordinarily recommended, the latter in a solution of 1,000 micrograms per cubic centimeter of sterile distilled water. The foam is placed on gauze to absorb some of the fluid so that it is not too wet, and then it is placed into the bone cavity (Fig. 324, *D*). The flap is returned over the foam without compressing it, and the wound closed by a sufficient number of sutures to prevent infection from entering. The vertical incision distal to the second molar must be given particular attention (Fig. 324, *E*). Kaldermic, dermalon, or other nonabsorbable sutures are best. A piece of dry gauze is placed between the jaws for half an hour. Cold applications to the cheek for twenty-four hours are recommended, and the sutures are removed after ten days.

The following operations serve as illustrations of the use of foam in space obliterations.

*Mr. R. B.*—July 14, 1944. Left lower third molar, class 4, unerupted, was excised. The tooth was bisected. The patient gagged, and saliva entered the wound. After removal of the tooth, the wound was treated with zephiran and stimulated to cause bleeding; foam soaked in thrombin and tyrothricin, equal parts, was inserted. Three sutures were placed.

July 17, 1944. There was practically no reaction. There was a small opening at the anterior part of the incision with no discharge.

July 24, 1944. The sutures were removed; the small hole healed by granulation.

August 14, 1944. Opening was well healed by granulation. Foam was not expelled.

*Mr. R. H.*—July 14, 1944. Right lower third molar, class 3, unerupted, was excised, after being bisected. Saliva entered the wound, which was treated with zephiran. Fibrin foam with thrombin and tyrothricin was inserted. Three sutures were placed.

\*Supplied through the courtesy of the Department of Physical Chemistry, Harvard Medical School, for experimental purposes.

†Supplied through the courtesy of Sharp & Dohme for experimental purposes.

July 25, 1944. Patient had some pain. Sutures were removed after ten days. Wound healed by first intention.

*Miss L. P.*—July 17, 1944. Cyst of right mandible was excised under avertin and ether anesthesia, with  $\frac{1}{100}$  grain atropine to arrest salivation. Foam, thrombin, and tyrothricin were placed in the wound and squashed down. Three sutures were placed. The recovery was uneventful. The foam was not expelled.

*Mrs. A. S.*—July 18, 1944. Right lower third molar, class 3, unerupted, and the left lower third molar, class 2 unerupted, were excised. Foam with thrombin and tyrothricin were placed into each wound, and closed with sutures.

July 25, 1944. Sutures were removed. Healing on the left was complete. On the right there was a small opening which healed by the formation of granulation tissue.

*Mr. W. H.*—Aug. 16, 1944. Left and right lower third molars excised; the operation was very difficult. Both were closed by sutures after inserting foam.

Aug. 17, 1944. Patient was fairly comfortable, but there was some post-operative edema.

Aug. 21, 1944. Wounds had healed by first intention, and sutures were removed.

*Master M. F.*—Aug. 23, 1944. Left and right lower third molars, partly developed and unerupted were excised. Both wounds were sutured after inserting foam.

Aug. 28, 1944. Sutures were removed. The recovery was uneventful.

*Miss P. G.*—Oct. 28, 1944. Excision of left and right lower third molars, class 4, unerupted. Foam was inserted as previously described. Wounds were closed with three sutures on each side.

Oct. 29, 1944. Considerable swelling of the face. No discharge.

Oct. 30, 1944. Sutures removed. Wound open on both sides; foam was expelled.

*Mr. T. LaR.*—Sept. 19, 1944. Excision of maxillary cyst. Cavity was filled with fibrin foam and closed by sutures. Wound healed without expelling foam.

*Mr. E. B.*—Jan. 16, 1945. Patient had Paget's disease (Fig. 400). An odontectomy was performed, and fibrin foam placed in the bone wound and closed with sutures. The wound healed by first intention without complications.

### THREE FRACTURES AT THE ANGLE OF THE JAW: ONE TREATED BY INTERNAL WIRING, TWO BY INTERNAL CLAMP FIXATION

KURT H. THOMA, D.M.D.

Skeletal fixation, although especially recommended for the edentulous mandible or edentulous sections of the jaw, has been found unsatisfactory in cases of fractures at the angle. Because the bone of the ramus is extremely thin, pins will not hold well. They are subject to much abuse at this place from pressure exerted when the patient rolls on the affected side while sleeping. These fractures, however, are particularly the ones for which we hoped skeletal fixation would be helpful, since they are generally associated with considerable displacement, due to muscle pull, and cannot be easily immobilized by inter-maxillary appliances. It is true that internal wiring fixation gives satisfactory results if the fracture is not septic, and if the mandible can be immobilized by wiring of the teeth. A case in which the fracture at the angle was treated in this manner will be presented. It may be inadvisable, however, to immobilize the jaw, or, as in the case mentioned, immobilization may be possible only by the construction of a splint, facilities for the making of which are not always available. In such cases skeletal fixation ought to be used, as it is the ideal method of treatment.

To overcome this handicap, I have experimented with a new kind of attachment and have developed a peripheral bone clamp that can be placed around the jaw through a suitable incision in the skin, which is closed, allowing only the pin to protrude through a separate stab incision. This clamp has three points of attachment: two sharp prongs engaging the inner surface of the bone, and a pointed pin which, when tightened, engages the outer surface of the jaw, at the same time fixing the inner prongs. A link of the Frac-Sure appliance is attached to the pin of the clamp, and a connecting rod is passed through its other hole. The other end of the rod is attached to the double link on the bar fixed on two half pins screwed into the anterior fragment at the mandibular border (Fig. 325).

The first model of the clamp which was used in the two cases to be described had a pin attached to the clamp which could not be removed because of a shoulder that prevented it from piercing the cortex of the bone (Fig. 326, A). This arrangement made the application of the clamp difficult. It had to be inserted by pushing the pin through the skin from beneath the flap and then placing the clamp into position.

The second model, which is also much less bulky because it is made of stainless steel, has a pin that can be inserted separately (Fig. 326, B). After the clamp has been seated without the pin, the site where the pin is to go is determined and incised by means of a stab with a No. 15 Bard-Parker blade. The pin has a thread the diameter of the shoulder of the clamp and this prevents it from entering the bone. Therefore, the incision must be a little larger than necessary for the part that projects through the skin. Such short incisions, however, heal easily and the skin closes tightly around the pin. The model



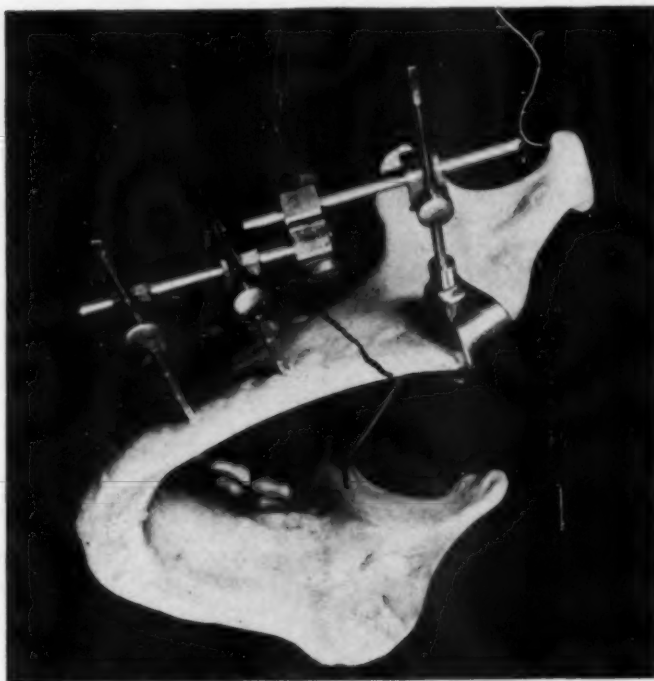


Fig. 325.—Fracture reduced by means of two pins in the anterior fragment and a peripheral bone clamp at the angle of the jaw.

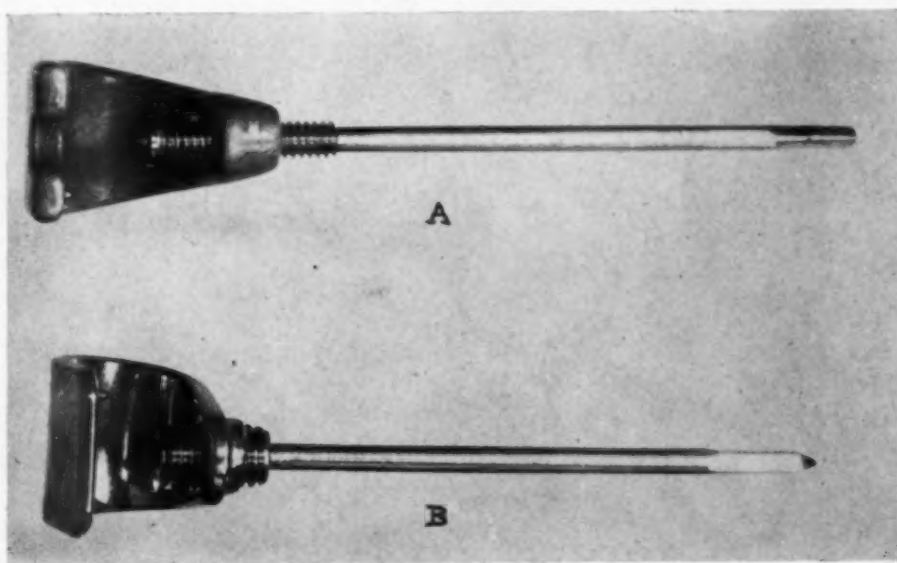


Fig. 326.—Peripheral bone clamp. A, First model; B, second model.

with the detachable pin also overcomes the difficulty which is encountered when removing the clamp. In the first model the pin had to be drawn into the wound in order to remove the appliance. Even if carefully sterilized the danger of drawing infection into the tissue was great. This risk is entirely eliminated by the second model, because the pin is removed first by unscrewing it, and then the clamp is taken out by incising the old scar.

The advantages of the internal clamp fixation are as follows:

1. The clamp can be attached to thin bone, and therefore is particularly useful at the angle of the jaw.

2. The clamp is attached to the cortices of the bone, and its use does not necessitate opening the spongiosa. It may therefore be used in septic fractures in which interosseous wiring and pin fixation are contraindicated.

3. The clamp does not cause bone resorption, which occurs frequently around the pins when they are subjected to stress. Therefore, clamps may remain longer than pins in this type of fracture fixation. This is important in fractures complicated by osteomyelitis (Case 73), and in bone grafting.

Two clamps may be used, one in each fragment, but this is not advocated because pins leave no scar, and can be inserted more rapidly without making an incision. The incision necessary to insert the clamp, however, if approximated by the subcuticular method, leaves a scar which is of minimal objection.

The case of a patient treated at an outside hospital is a good illustration of the advantage of clamp fixation over the pin fixation method. The patient had a double fracture of the mandible, one of which was located at the angle of the jaw and extended through an unerupted third molar. An attempt had been made to reduce and fix it by skeletal fixation. The pins in the ramus, however, could not be embedded sufficiently well to hold the displaced ramus in proper position. On account of this the appliance had to be removed. After treating the infection which had set in, with penicillin, 80,000 units daily intramuscularly, an open reduction was performed. After the third molar had been removed, the fracture was impacted under direct vision and fixed with a clamp placed at the angle of the jaw and two half pins which were inserted in the horizontal ramus. The postoperative x-ray showed a satisfactory position of the fragments.

Two other cases treated with internal clamp fixation will be described.

#### Case 71

##### **Fracture at Angle of Jaw Treated With Internal Wiring Fixation and Splint**

S. S. (291940), a 55-year-old man, came to the Dental Clinic Nov. 7, 1944, complaining of a swollen, painful jaw of five days' duration.

During an argument he was struck on the jaw five days previously, and was allegedly unconscious for two hours. The jaw was swollen and painful and he had severe headache and a watery discharge from the nose. His doctor referred him to a hospital where x-rays were taken and he was given pills for the headache. The x-rays revealed a fracture of the left mandible. On the day previous to entry here, he was discharged from the other hospital.

The patient had had a congenital neuromuscular disease involving the eye muscles (vertigo), hands (tremor), and tongue. Three years ago he had arthritis in the knee and interphalangeal joints. Since then he had had several



Fig. 327.—Fracture of jaw on left.

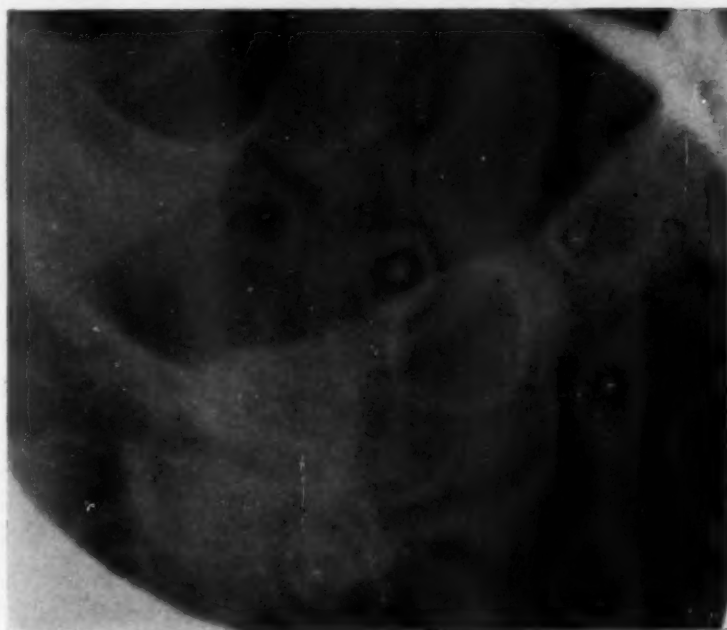


Fig. 328.—X-ray of mandible showing cyst on right.

attacks of less severity, and also numbness and tingling of the hands. His mother had congenital neuromuscular disease and also a brother and the patient's oldest son.

Examination revealed a swollen, tender left jaw and neck, with ecchymosis over the area and some edema of the mucous membrane. At present he had no pain nor headache. He seemed somewhat unresponsive and drowsy.

X-ray examination showed a complete transverse fracture at the angle of the left lower jaw with displacement of the fragments (Fig. 327). No other definite fracture could be seen. A cyst was visible in the right horizontal ramus of the lower jaw and was surrounded by a sharply defined margin of increased density (Fig. 328).

Diagnosis: Fracture of mandible on left.

The patient entered the House on November 10, and was kept under observation. In consultation with neurosurgery it was believed that he had had a head injury as he was disoriented and wild at times. There was a bloody tinge in the cerebral spinal fluid; the red blood cells were crenated; white cell count was 7,300, and hemoglobin 95 per cent.



Fig. 329.—Postoperative x-ray showing internal wiring fixation.

After a week the patient's condition had improved so that operation for fixation of the jaw could be performed on November 17.

Under endotracheal gas-oxygen-ether anesthesia, the skin was prepared in the usual way. An incision was made beneath the angle of the jaw on the left. The platysma was divided and the area of the fracture reached. After part of the attachment of the masseter muscle was removed the fracture came into view. The fragments were displaced and freely movable. The inner surface of the mandible was also stripped from its muscular attachment in the same area as on the outside. Then a hole was drilled into each fragment with an electric drill, the drill being cooled with sterile water while this was done. A stainless steel wire, 25 gauge, was inserted through the drill holes, the fracture was properly reduced, and immobilized by twisting the wire. The end was cut



short and bent over. Sulfanilamide powder was inserted. The subcutaneous tissue was closed with catgut and the skin with interrupted Kaldernie sutures.

The patient was given sulfadiazine postoperatively. On the next day a Gunning splint was inserted into the mouth, secured with a Barton bandage with elastic reinforcement. Postoperative x-rays showed the fragments held in good position (Fig. 329).

The patient was discharged on Nov. 22, 1944, to be seen in the Outpatient Department. He was seen several times in the Dental Clinic to have new bandages applied. The splints were removed on January 3, when the mandible was found to be firm. The patient was discharged to return in two weeks for excision of the cyst.



Fig. 330.—Fracture compounded into the mouth with displacement, complicated by a tooth in the fracture line.

#### **Case 72**

##### **Fracture at Angle of Jaw Treated With Internal Clamp and Pin Fixation**

M. C. (468071), an 18-year-old married woman, had fallen downstairs six days before and injured her jaw. There was slight bleeding, and she had been unable to close the jaws completely. She had had only fluids since the accident. Antitetanus serum was given.

Examination revealed that the mandible was fractured and displaced to the left. X-ray examination showed a fracture of the left mandible through the region of the remaining molar tooth (Fig. 330). There was no fracture on the right side, nor of the condyles.

On Oct. 20, 1944, the patient entered the hospital and was given chemotherapy. On the next day, under intratracheal gas-oxygen-ether anesthesia, an incision 3 cm. in length was made 1 cm. below the angle of the jaw. The platysma was divided, and by blunt dissection the bone of the mandible was reached. A few small vessels were tied, and two retractors were inserted. Part of the masseter and internal pterygoid muscles were stripped from an area at the angle of the jaw. The clamp was then inserted, and after a stab incision for the pin was made further up in the skin, it was fixed to the bone by fastening the pin with the wrench (Fig. 331). The subcutaneous tissue was closed over the clamp with catgut sutures (Fig. 332), and the skin with interrupted dermalon sutures. Two half pins were next inserted into the anterior fragment near the lower border of the jaw in the usual manner by drilling holes just through both cortices with a spear-shaped bur. The Frae-Sure appliance was next assembled, but not as yet permanently adjusted. The incisions around the pins were painted with collodion, and dry dressings were applied, as shown in Fig. 333. Next, the mouth was opened with a mouth gag and the mucosa prepared with zephiran. The molar in the fracture line was removed with forceps, and sulfanilamide powder was placed in the tooth socket. The mouth prop was removed and the fracture reduced by manipulation. When it was satisfactorily aligned, it was immobilized by the assistant who fastened the nuts of the connecting rod of the appliance. A gauze pack was placed over the socket and held in position with a Barton bandage.

The postoperative x-ray examination showed the previously described fracture maintained in good position and alignment by the external fixation apparatus (Fig. 334).

On October 24 the patient's temperature rose to 103.8° F. She had practically no swelling of the jaw, however, no pain or other discomfort. There was a fine "measly" rash over the whole body. The posterior cervical nodes were enlarged but not tender. In consultation it was thought that the patient might have German measles, but she said she had had them, so it was decided that the rash and fever were due to the sulfadiazine therapy. The sulfadiazine was stopped and blood and urine examinations done. The urine examination was as follows: cloudy, amber, acid, 1.020 specific gravity, some albumin, 3 red blood cells, 1.5 white blood cells, and 20 epithelial cells. The white cell count was 8,300. On the next day the white cell count was 9,000, 65 per cent polymorphonuclear leucocytes, 2 per cent large lymphocytes, 18 per cent small lymphocytes, 5 per cent mononuclear cells, 7 per cent eosinophiles, and 1 per cent basophiles. The Hinton test was negative.

Two days after the sulfadiazine was stopped the temperature returned to normal and the rash disappeared. The patient was discharged on Oct. 28, 1944, to be followed at the office.

Five weeks after the operation the skin condition was very satisfactory. The patient had only a slight amount of seepage around the pins. There was absolutely no reaction from the clamp, no tenderness on palpation, and it seemed to be as firmly attached as the day it was applied (Fig. 335). The connecting bar was removed to test the fracture, and it was found to be firmly united. It



Fig. 331.—Insertion of the peripheral clamp.

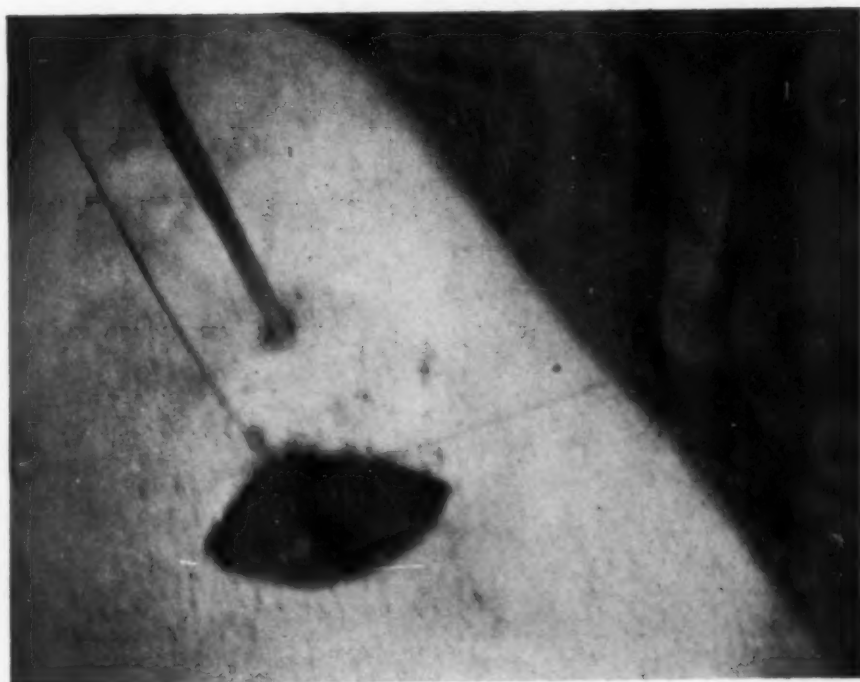


Fig. 332.—Closing the subcuticular tissue over the clamp.

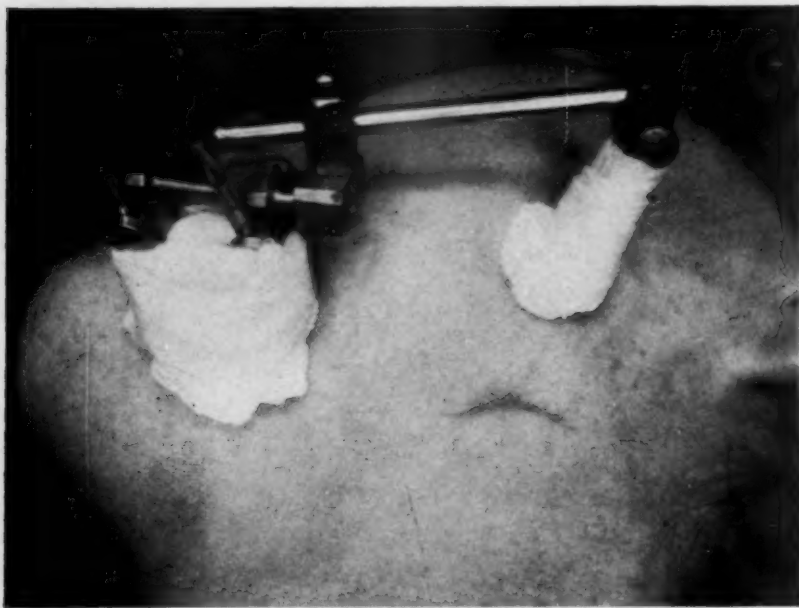


Fig. 333.—Clamp and pin fixation inserted and fracture immobilized by connecting bar. Photograph taken three weeks after operation.

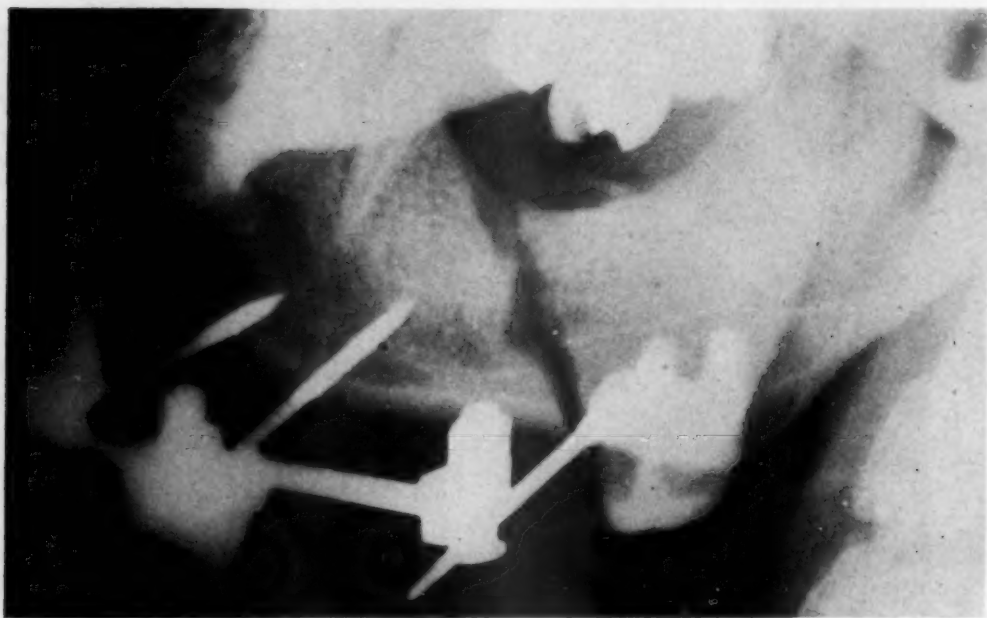


Fig. 334.—X-ray showing clamp and pin fixation after fracture has been reduced.



was decided to remove the appliance the next day under a short intravenous pentothal sodium anesthesia in the Outpatient Department. The half pins were taken out, and an incision, including removal of the old scar, was made. Tissue was dissected down to the clamp which was removed. The subcutaneous tissue was then carefully approximated and closed by suture; the skin incision was closed in a similar manner. The incisions where the pins protruded through the skin were treated by injecting zephiran, and a small dressing was applied.

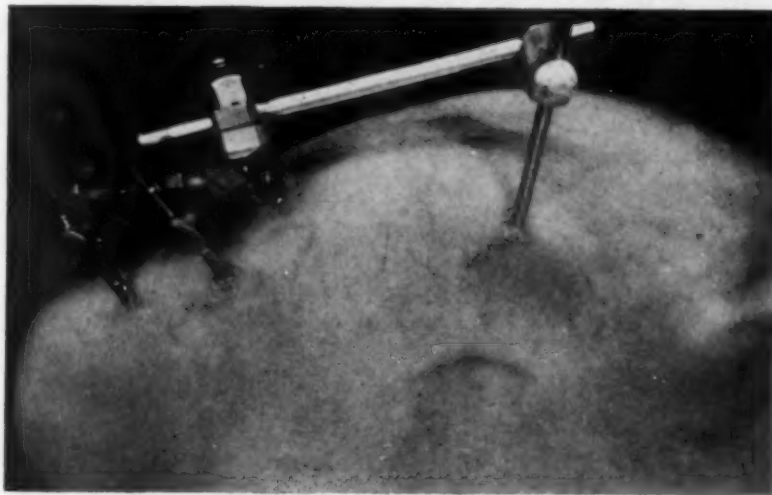


Fig. 335.—Photograph taken five weeks after operation to show condition of puncture wounds and incision. There was only a very slight amount of seepage, and practically no reaction.

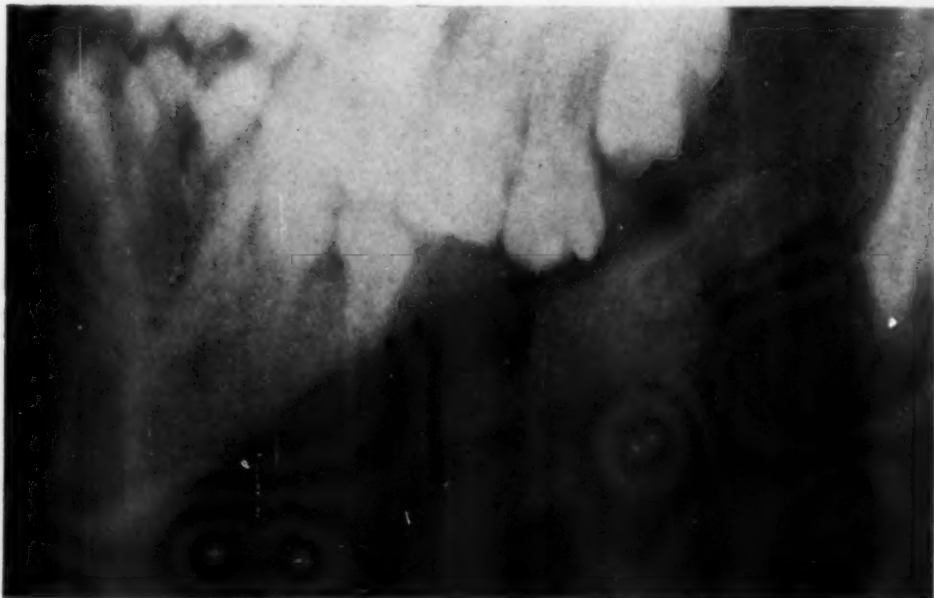


Fig. 336.—X-ray taken after the removal of appliance when good union had occurred.

An x-ray taken two days after removal of the appliance (Fig. 336) showed that no bone resorption had occurred at the part of the jaw where the clamp had been attached.

## Case No. 73

**Septic Fracture at Angle of Jaw With Osteomyelitis,  
Treated by Internal Clamp and Pin Fixation**

V. T. (468102), a 47-year-old man, presented himself at the Outpatient Dental Clinic on Oct. 19, 1944, complaining of a swelling on the right mandible. Two months previously, during a fist fight, the patient had been struck on the jaw; he did not lose consciousness and walked to his home later. The right jaw became swollen, slightly tender, and painful, but he was able to move the jaw enough to eat.

Examination revealed a large swelling about the size of an apple on the outside of the face (Fig. 337). This mass was fluctuant, and probably was a submaxillary abscess containing pus. Examination of the mouth showed that he had lost many teeth; there were no teeth present from the lower right canine region to the third molar region. There was mobility of the jaw on the right, indicating fracture.

X-ray examination showed a fracture of the horizontal ramus of the right mandible close to the angle, with some eburnation of the adjacent bone. There had been considerable decalcification along the fracture line, probably due to bone resorption. The reaction of the bone adjacent to the fracture indicated that there was some inflammatory process involving the mandible (Fig. 338).

**Diagnosis:** Fracture of the mandible with osteomyelitis.

The patient was admitted to the House on October 23, and started on penicillin, 10,000 units intramuscularly, every three hours. The abscess was incised under local anesthesia, and about four ounces of pus evacuated. A rubber dam drain was inserted and attached to the skin with a suture.

A culture taken from the pus showed moderate nonhemolytic streptococci on a blood agar plate, and alpha hemolytic streptococci on plain broth. The Wassermann test was negative, but the Hinton was positive. The blood count showed red blood cells, 4,850,000; white blood cells, 8,700; and hemoglobin, 75 per cent. A culture from the draining sinus two days after incision showed *Staphylococcus aureus*.

On October 27, after the usual premedication and under gas-oxygen-ether anesthesia, the incision which was made a few days previously was enlarged, and the inferior border of the mandible exposed. No pus was encountered, but there was some serous discharge. The fracture was exposed and it was found that while a large area of the outer cortex had been destroyed by infection, the inner cortex had not been affected, and the fragments here were in contact. The area that gave the appearance of a distracted fracture, therefore, was produced by the destruction of the outer cortex of the bone. The bone was carefully débrided and all pathologic tissue removed. Since there was practically no pus encountered and very little swelling remained, it was decided to apply skeletal fixation at this time, although it had been planned to defer using it until a few days later.

The angle of the jaw was exposed through the incision, and muscle was detached on both the inner and outer sides. The clamp was then fitted on. The pin was pushed through a stab incision in the skin and the clamp fastened



Fig. 337.—Septic fracture at the angle of the jaw with submaxillary abscess.

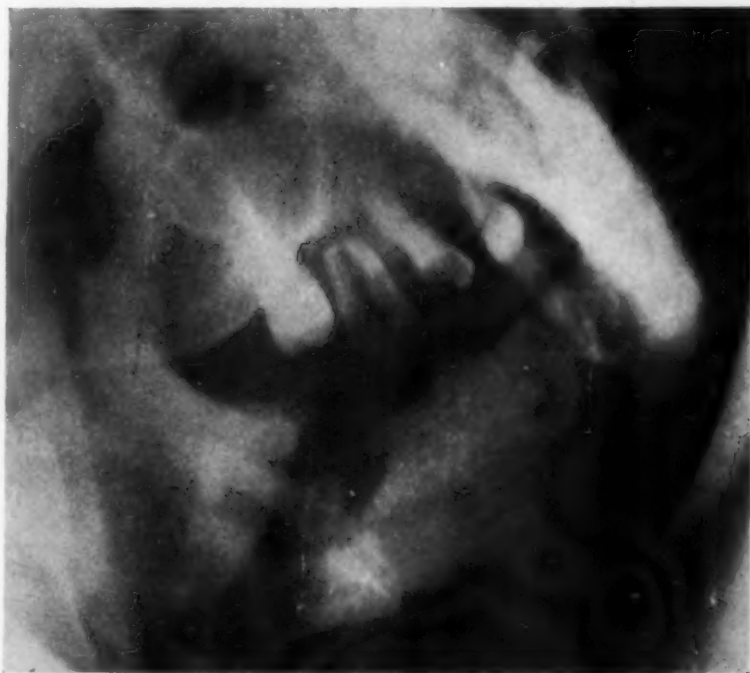


Fig. 338.—X-ray showing fracture with osteomyelitis.

to the bone. This gave a firm hold to the posterior edentulous fragment. Two pins were inserted with a hand drill in the anterior fragment away from the infection, in the inferior border of the mandible through the skin. The fracture was then manipulated and properly impacted, and the crossbars of the



Fig. 339.—Photograph showing clamp and pin fixation to immobilize the fracture after reduction.

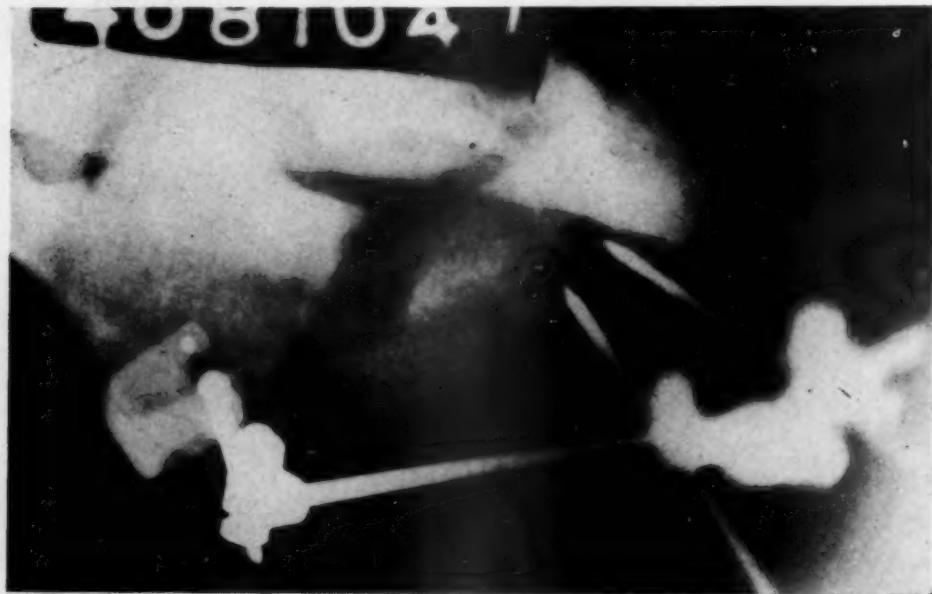


Fig. 340.—Postoperative x-ray showing clamp attached at the posterior border of the ramus. Pins were inserted in the anterior part of the mandible. Fracture was impacted at the inner plate; some of the outer plate had been destroyed by osteomyelitis.



Frac-Sure appliance were attached to fix and immobilize the fracture. A Dakin tube was placed into the wound. To prevent it from being displaced, it was sutured with silk to the skin, and the incision was closed. Colloidon was painted on the perforation in the skin around the pins, and a dry dressing applied (Fig. 339).

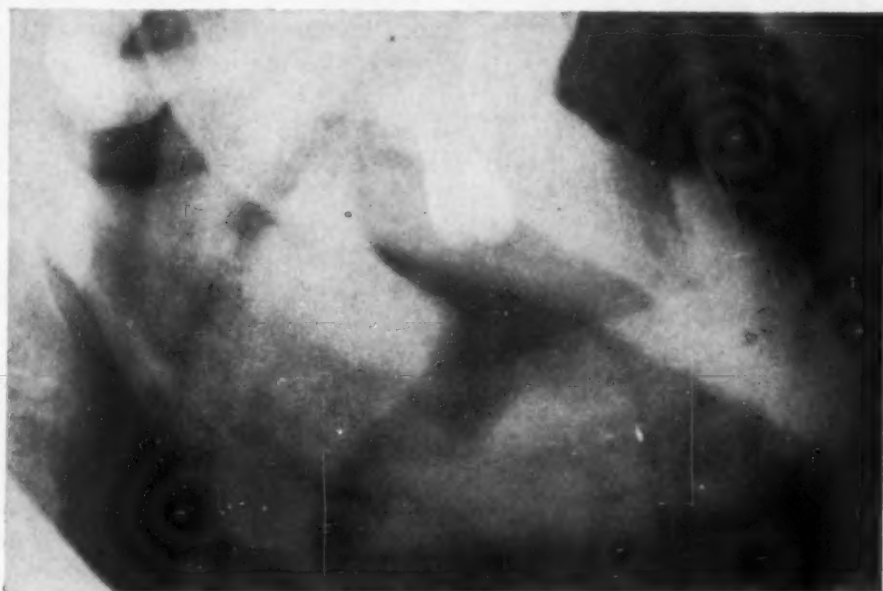


Fig. 341.—X-ray taken after removal of the appliance, five and one-half weeks after reduction, with good union of the jaw at the lingual side.

The penicillin was continued postoperatively, 10,000 units intramuscularly, and 250 units per c.c. was locally instilled into the tube in the wound every three hours. On October 31 the patient was discharged to the Outpatient Department, because he was very restless and wanted to go home. This proved to be a mistake, however, because when he was next seen the following week, a small amount of pus was still draining from the fistula. The wound was irrigated with Dobell's solution daily but continued to drain. X-rays taken on November 16 showed that the fragments were maintained in good position by the splint (Fig. 340). On November 27 the jaw was manipulated and it was found that considerable union had taken place. A few drops of pus exuded from the sinus, however, and since a small sequestrum was thought to be seen in the x-ray taken previously, the patient was readmitted to the House for sequestrectomy and removal of the splint. On November 30, under endotracheal gas-oxygen-ether anesthesia, an incision was made at the angle of the jaw. It included the fistula which was excised. Two sequestra were encountered and removed. Then the clamp was located, and after the pin was loosened and sterilized, the apparatus was removed. The bone was carefully inspected and an infected area was discovered containing considerable granulation tissue, which was completely excised. One gram of sulfadiazine powder was dusted into the area and over the exposed bone; a rubber dam drain was placed in the wound and the subcutaneous tissue closed with catgut. The skin was sutured with interrupted dermalon sutures. The two pins which had been inserted into

the anterior part of the horizontal ramus were removed also, and zephiran was injected into the perforations in the skin and the holes in the jaw. A dry dressing was applied.

The patient was given sulfadiazine postoperatively for six days, 3 Gm. the first day and 6 Gm. a day thereafter. The culture from the pus showed *Staphylococcus aureus* and nonhemolytic streptococci. The white blood count was 8,050 and hemoglobin 76 per cent. On December 4 the x-ray report showed that the splint had been removed. The bone appeared rather dense; there was no evidence of resorption caused by the clamp (Fig. 341).

The wound was irrigated daily, and the patient was discharged on December 9 to the Outpatient Department. He was last seen on December 20 when the fistula was closed and the jaw was solidly united.

### FRACTURES OF THE CONDYLE OF THE JAW

KURT H. THOMA, D.M.D., HENRY D. HOWE, D.M.D., AND MARTIN WENIG, D.D.S.

In a recent paper\* the subject of condylar fractures was presented in considerable detail, together with a new technique for reduction and fixation when displaced or dislocated. During the past two years, thirty-five condylar fractures were treated in this clinic. Table I is an analysis of these fractures.

TABLE I  
ANALYSIS OF 35 CASES OF CONDYLAR FRACTURES

Number of fractures	35	Number of patients	29
Condylar	30	Males	20
Subcondylar	5	Females	9
Right condyle	22	Unilateral	23
Left condyle	13	Bilateral	6
No displacement of condyle	4	No other mandibular fractures	12
Displacement laterally	7	One other mandibular fracture	12
Displacement medially	4	Two other mandibular fractures	2
Comminuted	2	Comminuted mandibular fracture	3
Fracture dislocations, medially	10	Treated by intermaxillary fixation	25
Subluxation	4	With open reduction without	
Complete dislocation	3	local fixation	1
Complete dislodgement	2	With open reduction and internal	
Dislocation of part of condyle	1	wiring fixation	7
Fracture dislocations forward	1	With open reduction and	
Fracture dislocations with displaced		skeletal fixation	5
meniscus	1	With open reduction, internal	
Old fracture with pseudarthrosis	1	wiring, and skeletal fixation	1
With ankylosis	3	Condylectomy (fresh cases)	1
		Osteoarthrotomy (old cases	
		with ankylosis)	4

As a result of a careful study of these cases, the following deductions were made:

1. The contention of most writers that conservative treatment gives consistently good results is not in accord with our findings. Contrarily, we have seen patients who came to us with both major functional disturbances, such as

\*Thoma, K. H.: J. Oral Surg. 3: 3, 1945.

pseudarthrosis or ankylosis, as well as minor complaints such as pain, limited motion, and malocclusion, resulting from malunion after intermaxillary fixation by wiring or elastic traction.

2. We were able to observe that, in children, spontaneous readjustment of the mandibular joint due to epiphyseal growth of the condyle, and positioning of the teeth, will, in time, greatly aid in the correction of minor defects following treatment. This is especially true of malocclusion. In adults, however, and particularly in cases in which the condylar fragment is displaced, dislocated, or comminuted, better results can be obtained by operative procedures.

3. We feel that with open reduction and internal wiring fixation, greatly improved results may be obtained over those gained by conservative treatment. This is especially true in cases in which there is wide separation of the fragments disposing to nonunion, or marked overriding and displacement of the condyle which would produce shortening of the ramus and open-bite, or mechanical interference with proper functioning of the joint.

4. For the reduction of fracture dislocations we feel that, in addition to internal wiring fixation, immobilization of the condyle in the glenoid fossa by skeletal fixation, at least for a short period of time, gives greater security in holding the condyle in position, preventing it from being redislocated while the mandible is positioned and immobilized, during recovery from anesthesia, or when the other side is subjected to operation.

5. Immobilization of the mandible until there is sufficient union of the condylar fracture is required even though internal wiring is used. It is important, however, to start functional treatment of the joint as early as possible, particularly in fracture dislocations. The movements of the jaw should be restricted at first by intermaxillary elastics, later aided by finger pressure.

6. The proper handling of concurrent fractures in the horizontal ramus is important. Since functional treatment to re-establish normal use of the joint should be initiated before such fractures have completely healed, their fixation must not depend on prolonged intermaxillary wiring; they should be fixed by skeletal fixation or any other method which allows mobilization of the mandible when required.

A case of bilateral fracture of the mandibular condyles, complicated by fracture at the symphysis, will be presented. This case is a good illustration of the difficulties and complications that may be encountered in this type of injury. It shows the application of the internal wiring method on one side, and on the other, the use of internal wiring combined with skeletal fixation to insure the reduced dislocation from being dislodged again.

#### **Case 74**

##### **Bilateral Fracture of the Mandibular Condyles, With Fracture at the Mandibular Symphysis, Treated by Internal Wiring and Skeletal Fixation**

R. F. (464422), a 42-year-old man, was brought to the Emergency Ward after an automobile accident in which he struck his chin on the windshield. He did not lose consciousness, and there were no other injuries.

On examination the patient was found to be suffering from (1) a fracture of the mandible to the right of the symphysis between the right first and second incisors with displacement, and compounded into the mouth; (2) fracture dislocation of the right condyle, dislocated about 80 degrees medially with contact of the fragments; (3) fracture of the base of the neck of the left condyle with displacement (Figs. 342 and 343, *A* and *B*); and (4) an open-bite due to impaction of the condylar fractures and spasms of the elevator muscles.

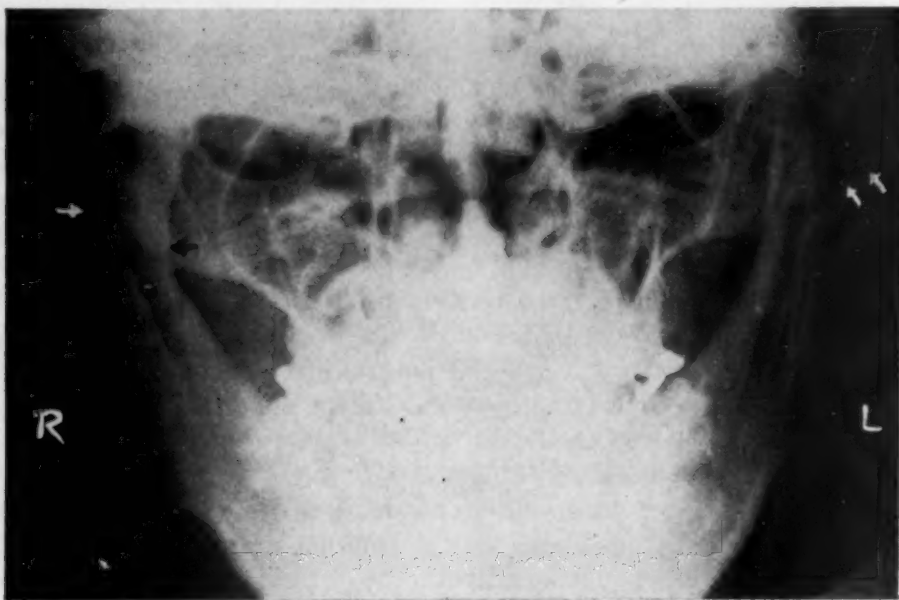


Fig. 342.—Anteroposterior x-ray showing bilateral fracture of the mandibular condyles with displacement on the left and fracture dislocation on the right.



*A.*

*B.*

Fig. 343.—*A*, Lateral view of fractured condyle on the right; *B*, on the left.

A temporary reduction with horizontal wiring of the anterior fracture was done and a Barton bandage applied. The next day, Jelenko splints were wired to the upper and lower jaws, and intermaxillary elastics were applied in the



anterior part of the mouth to close the open-bite and bring the teeth into occlusion (Fig. 344). The open-bite was improved by this treatment, but not entirely relieved after forty-eight hours (Fig. 345). This was believed to be due to the excessive elevation of the ramus made possible by the bilateral condylar fractures and dislocation. Open reduction, it was felt, would give the most satisfactory result.

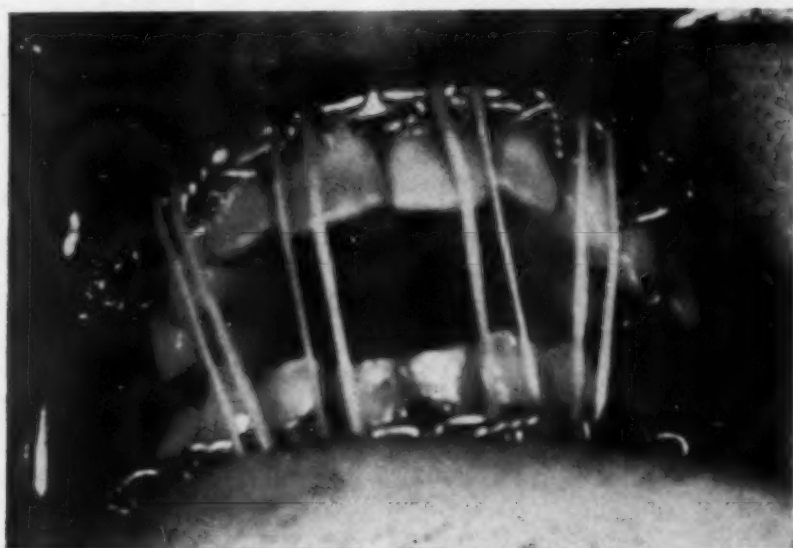


Fig. 344.—Open incisor bite caused by condylar fracture, immediately after applying elastic traction on Jelenko splints.



Fig. 345.—Improvement in bite after forty-eight hours, but normal occlusion could not be obtained.

Two days later, therefore, an open reduction of the fracture dislocation of the right condyle was performed. After the usual premedication and under pentothal sodium anesthesia, an angulated vertical incision was made in front of the right ear extending to the attachment of the lobe. The subcutaneous

tissue was divided, and the superficial orbital arteries and veins cut and ligated. The zygomatic arch was exposed and the joint cavity was found to be empty. The capsule and the temporomandibular ligament were intact on the external side. The neck of the condyle was exposed and found to be fractured at the base obliquely across from the mandibular notch to the posterior border of the ramus. The condylar fragment was found completely detached from the capsule, but it was attached to the external pterygoid muscle by a few muscle fibers. While it was held with bone forceps, an oblique hole was drilled into the condylar fragment from the external surface to the fracture surface. Then the condyle was placed medially by means of a bone file, and, by pressing on the chin, the mandibular fragment was brought into the incision. A hole was drilled here also, again from the external surface to the fracture surface. A sterile stainless steel wire, 25 gauge, was inserted through the two holes, and after the fracture was properly impacted, the wire was tightened, cut, and bent. The head of the condyle was then pulled into the glenoid fossa by means of hooks. While it was held in position with retractors, a hole was drilled into its lateral surface at the junction of the head and the neck and a half pin inserted. Another half pin was drilled into the eminentia articularis, through a stab incision in the skin. The capsule was sutured with catgut. After the wound was closed, the condyle was positioned and a Frac-Sure bar attached in such a manner that the condyle was pulled in an outward direction and thus completely fixed in its reduced position. A dressing and an elastic Barton bandage were applied.

Four days after the first operation, the second operation was performed on the left, with the same type of anesthesia and skin preparation. The condyle was found displaced medially as well as forward. The mandibular fragment came into view when the chin was pressed back, and a hole was drilled through the fractured end for the insertion of a stainless steel wire, 25 gauge. The condylar fragment was located and, by means of a bone file, moved from the medial side to the external side of the ramus while its head was held with bone forceps. A hole was drilled through the neck of the condyle and a double wire inserted, by means of which the wire in the other fragment was drawn through. The condyle was then impacted, the fragments were placed in good position, the wire was made taut, twisted, and cut off, and the wound was closed. A dry dressing was applied to the face around the pins. The occlusion was locked by placing a brass wire around two lugs of the Jelenko splints, one on each side. Post-operative x-rays are shown in Fig. 346.

The day after the second operation there was some discharge around one of the pins inserted at the first operation. The culture showed the presence of hemolytic *Staphylococcus aureus*, 3 plus sensitive to penicillin. The patient was immediately placed on penicillin; 10,000 units were given intramuscularly every three hours, and, since the pins had served their purpose, they were removed (sixth day after being inserted). Lateral x-rays taken at this time are shown in Figs. 347 and 348. One to two cubic centimeters of a solution of 500 units per cubic centimeter of penicillin was injected into the wound every three hours through the pin holes in the skin. There was some discharge for three days, and a little swelling which disappeared after seven days. Otherwise the

patient progressed satisfactorily and was discharged eight days after the second operation, to be followed at the office. Four weeks after the second operation the intermaxillary wires were removed and the patient was allowed some motion restricted by two elastics. After another week the Jelenko splints were removed.



Fig. 346.—Postoperative x-ray showing internal wiring fixation on one side and interosseous wire fixation with skeletal fixation on the other.

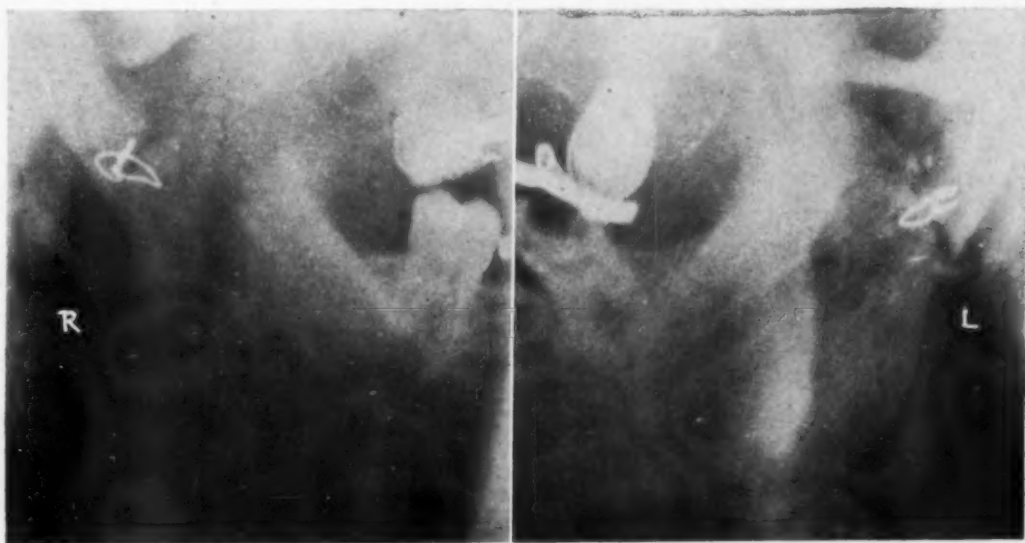


Fig. 347.

Fig. 348.

Figs. 347 and 348.—Postoperative x-rays showing wiring of fracture in lateral view.

The mandibular fracture at this time was firmly united, but the patient had slight muscular spasm which limited the motions of his jaw. With exercise, this condition, however, subsided promptly.

*Discussion.*—The clinical result in this case was excellent. The reduction, however, was not anatomically perfect as it has been in some of the other cases wired by this method. In the x-ray (Fig. 346) an angulation of the condyles is revealed which is not due to medial dislocation but to a spreading of the jaw. The reason for this spreading is the anterior fracture, which, when present, acts as a hinge allowing the rami to diverge; this may not be easily noticed if the patient has poor occlusion. This divergence does not occur when there is no fracture in the horizontal rami. The accident as well as the operative procedure tends to displace the fragments outward, and, therefore, in fractures like the one described, one should take care to push the wired fragments medially before closing the wound. Perhaps putting some spring into the Jelenko arch attached to the mandible might help to press the rami together.

### FRACTURES OF THE MIDDLE THIRD OF THE FACE

KURT H. THOMA, D.M.D., HENRY D. HOWE, D.M.D., AND MARTIN WENIG, D.D.S.

Among the fractures seen during the past six months, there were four which involved the middle third of the face. In one instance the fracture involved only the zygomatic arch; in two the zygoma or malar bone was fractured and impacted in the maxillary sinus. In all three cases the injury was caused by a direct blow to the affected part of the face.

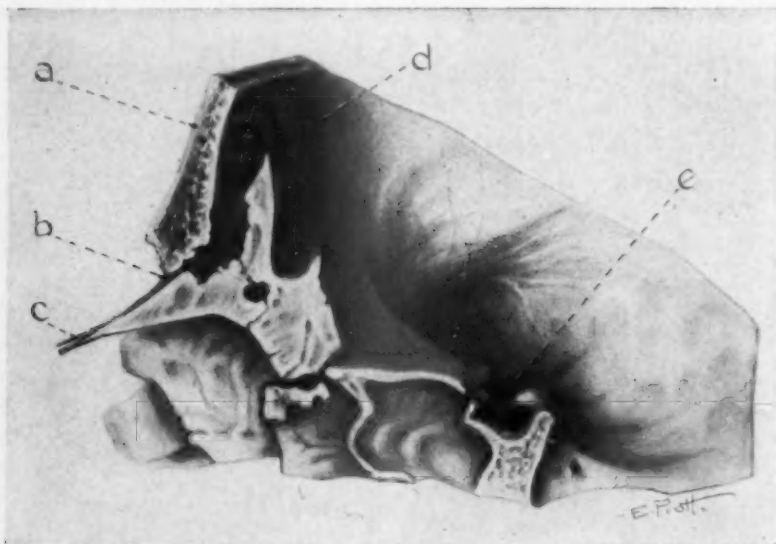


Fig. 349.—Sagittal section of skull showing fracture of the middle third of the face. *a*, Frontal bone; *b*, line of fracture; *c*, nasal bone; *d*, anterior cranial fossa; *e*, sella turcica.

One fracture was very extensive; the patient had been unconscious and later irrational. She had cerebrospinal rhinorrhea, and was blind in one eye, the eyeball having been punctured. This again is one of those very serious injuries, of which two examples were published in *Volume II* (Cases 30 and 31). The death rate in these cases is extremely high.



In extensive injuries of the middle third of the face, the bony facial structure generally is pushed into the anterior cranial fossa and impacted under the projecting edge of the frontal bone (Fig. 349). Reduction is usually accomplished by traction, since, in such patients, the fracture must not be manipulated until the fractured cribriform plate from which the cerebrospinal fluid leaks has been repaired. In contradiction to the advice of many writers, the reduction of these fractures does not require an upward pull, like that produced by the reversed Kingsley splint, but a down and forward pull to disimpact the facial bones and gradually bring them into their anatomical relationship. "Bird-cage traction," applied by means of a rubber band attached to a Jelenko arch on the upper jaw, and stretched to a coat-hanger apparatus extended from a plaster cap, has been found helpful in these cases. If the maxilla is separated from its superstructure by a horizontal fracture, traction can be applied by the use of eyescrews which are inserted through the skin into the malar bone.

### Case 75

#### Fracture of the Zygomatic Arch

A. B. (465472), a 73-year-old man, was brought to the Emergency Ward on Oct. 1, 1944, after having been involved in an automobile accident. He sustained a blow to the left cheek bone when his companion's head hit against his face. He did not lose consciousness; he had no nosebleed, stiff neck, or headache. There was no trauma to the chest, abdomen, or back. The past history revealed that the patient had sustained a depressed fracture of the right zygoma many years ago.

Examination showed a definite depression in the cheek with redness and edema, and marked tenderness. There was a superficial laceration of the scalp over the occiput.

Diagnosis: depression fracture of the left zygoma. The blood examination showed a white cell count of 7,000, hemoglobin 13.6 Gm., and nonprotein nitrogen 29.0 mg. per liter.

X-ray examination revealed a depressed fracture of the left zygomatic arch with disarticulation at the zygomatic temporal suture (Fig. 350).

Chemotherapy was instituted on October 1, and the next day, under intra-tracheal gas-oxygen-ether anesthesia, a reduction of the fracture was performed. After the usual preparation of the oral mucosa with tincture of zephiran, an incision was made on the left side of the maxilla behind the zygomatic process. With the mouth opened wide by means of a mouth gag, a curved periosteal elevator with a blunt edge was inserted and passed up under the inner surface of the zygomatic arch. One finger was placed under the instrument to form a fulcrum, and the depressed bone was pushed outward. It could be felt to snap into place by the palpating finger placed into the depression of the cheek. The wound was closed by suture.

The postoperative course was uneventful. A postoperative x-ray (Fig. 351) showed the fracture satisfactorily reduced. The patient was discharged on Oct. 6, 1944, relieved.



Fig. 350.—Fracture of the zygomatic arch.



Fig. 351.—Fracture of the zygomatic arch reduced.

**Case 76****Fracture of the Zygoma**

H. N. (342781), a 27-year-old man, came to the Emergency Ward on Oct. 8, 1944, complaining of numbness and swelling below the left eye. Three days previously he was struck over the left cheek by a fellow worker. He had suffered no pain then nor since. After the blow, however, he noticed numbness of the left side of the nose and upper lip. The cheek became swollen and discolored. X-rays taken at another hospital showed a fracture of the facial bones, and the patient was referred to our clinic.

Past history revealed that the patient had contracted a compound fracture above the left ankle twelve years before, and a compound fracture of the left frontal bone fourteen years before.



Fig. 352.—Fracture of the maxilla and malar bone.

Examination showed an asymmetrical face; the left side failed to participate in grimaces and the patient could not whistle. The left side of the nose and cheek was greatly swollen, and sensation over this area was impaired both in regard to light touch and pain. Marked ecchymosis was noted about the left eye. The white blood count was 7,000 and the hemoglobin 80 per cent.

X-ray examination on October 9 showed that there was a fracture of the left zygoma with slight displacement. There was a break at the infraorbital margin, and two fractures were demonstrated in the lateral wall and roof of the left maxillary sinus (Fig. 352).

Sulfadiazine therapy was instituted, and, on October 10, under intra-tracheal gas-oxygen-ether anesthesia, open reduction of the fracture was undertaken. An incision was made in the canine fossa. After the mucoperiosteum was stripped from the bone, the fracture became visible. It extended vertically and horizontally through the outer wall of the maxilla. With a gouge a small hole was made in the canine fossa, and by means of backbiting forceps, this hole was enlarged until it was of sufficient size to decompress the fractures. A urethral sound was inserted, and with this the malar bone was pushed out so as to improve the displacement at the infraorbital margin, and the frontozygomatic suture. An opening was then made from the nose to the maxillary sinus by means of a trocar and rasp, inserted through the nostril. A petrolatum Mosher strip was placed into the maxillary sinus to hold the decompressed fragment of the outer wall of the sinus in position. The end was pushed through the nasointral window into the nose, and allowed to protrude from the nostril, where it was attached to the skin with a piece of tape. The incision was closed with interrupted silk sutures.

Sulfadiazine therapy was continued. On the first postoperative day there was considerable swelling of the tissues and ecchymosis about the left eye. Cold packs were applied. On October 14 there was still some swelling present. The Mosher strip was removed. Postoperative x-rays showed satisfactory results of the operation, and the patient made an uneventful recovery. He was discharged to the Outpatient Department on Oct. 18, 1944.

When the patient was seen again on October 25, the fracture seemed to be healed. Except for discharge of blood from the nose at one time, there had been no nasal complications. He did complain, however, of occasional pain on the left side of the face referred to the eye, and also in the upper lip and several teeth. This was due to the fracture line which extended obliquely down from the internal canthus of the eye to the first molar area.

The patient was seen again on December 6; he had progressed satisfactorily, was relieved of the pain, and was permanently discharged.

#### **Case 77**

#### **Fracture of the Middle Third of the Face Treated With Bird-Cage Traction**

A. K. (464866), a 17-year-old girl, was brought to the Eye and Ear Infirmary from another hospital on Sept. 24, 1944, after having been in an automobile accident. She had been unconscious, and after regaining consciousness was irrational. After consultation with the neurosurgical, nose and throat, and surgical services, she was admitted to the general hospital.

Examination revealed that she was suffering from a punctured left eyeball, contusion of the eyelids, a broken nose, lacerations of the face and extremities. Cerebrospinal fluid was running from the nose, which was depressed (Fig. 353). She could not approximate the teeth, nor open the mouth more than  $1\frac{1}{4}$  inches. Impression: fracture of the cribriform plate, nasal and malar bones, dislocation of the mandible, fractures of the wrist, and punctured eyeball. Blood examination was as follows: white cell count, 14,700; red cell count, 4,480,000;



hemoglobin, 70.5 per cent; blood sugar, 100 mg. per liter; protein, 5.8 mg. per liter. She received a high protein, carbohydrate, and vitamin, liquid diet.

The patient was given sulfadiazine, 2 Gm. to start and 1 Gm. every three hours, and penicillin, 12,000 units every three hours intramuscularly. On October 2 her condition had improved considerably. The rhinorrhea had stopped, and it was advised that she have enucleation of the left eye and repair of the fractures of the face. On October 4 enucleation of the eye was carried out.



Fig. 353.—Patient with middle-third face fracture sustained in automobile accident.

The x-ray examination which was made in the meantime showed multiple fractures of the facial bones. There was a depressed fracture extending into the frontal sinus and probably involving the cribriform plate (Fig. 354). The left zygoma was probably fractured, and the left orbit appeared to be encroached upon by the fractured fragments. X-rays of the sinuses revealed multiple fractures of the facial bones involving the nasal bones, ethmoids, and both maxillary and inferior portions of the malar bones (Fig. 355). No fracture of the mandible was seen.

On Oct. 13, 1944, reduction of the middle face fractures was performed. An intravenous setup was connected to the patient's arm in order to inject a small amount of pentothal sodium when needed at times when the operative procedures became painful. Jelenko splints were fitted to the upper and lower jaws and attached with stainless steel wires to the teeth. Intermaxillary elastics were applied between the splints in order to reduce the middle face fracture gradually by bringing the maxilla and its suprastructures down and forward



**Fig. 354.**—Lateral head x-ray showing fracture of middle third of face, and involvement of the maxilla, ethmoid, and cribriform plate.



**Fig. 355.**—Anteroposterior x-ray showing fracture of malar bone and distortion of outline of the orbit.

to occlude the teeth (Fig. 356). Bird-cage traction was applied by attaching an elastic band by means of a wire to the upper Jelenko arch in the center, and the other end to a previously prepared "coat-hanger" apparatus incorporated into a plaster cap (Fig. 357). The patient was returned to the ward in good condition. She was given an intravenous infusion of 1,500 c.c. of 5 per cent glucose and saline.

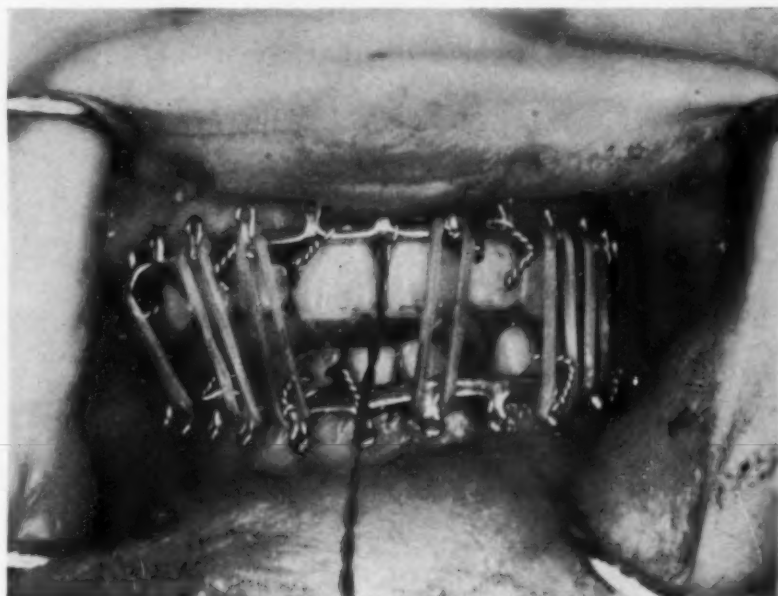


Fig. 356.—Jelenko splints applied with intermaxillary traction.



Fig. 357.—Plaster cap with bird cage arrangement for additional external traction.

Penicillin therapy was continued until October 26. She was given vitamin B complex and vitamin C. Twenty-four hours after the operation the teeth were found to be in occlusion, and two days later rubber-dam traction was applied under the chin to close the frontomalar sutures (Fig. 358). The patient's progress was satisfactory, and she was discharged from the House on Nov. 3, 1944, to be followed in the Outpatient Department.



Fig. 358.—Fracture has been reduced by traction and teeth are in occlusion.

The patient's progress was found to be good on November 22. The inter-maxillary fixation was removed to test the fracture. Pressure applied at various places caused no pain, but there seemed to be very slight mobility in the fracture between the malar and frontal bones. The Jelenko splints were removed. The patient had good occlusion and was advised to masticate soft foods. One month later there was no more mobility of the fractured bones, and the patient was referred to the Plastic Clinic for restoration of the nose.



## OSTEOMYELITIS

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Three cases of osteomyelitis and one case of tuberculosis are presented in this issue. One case of osteomyelitis developed a sequestrum at the inferior border of the mandible from an infection on a tooth. No doubt the osteomyelitis developed because the original treatment of the submaxillary abscess had not included the removal of the cause, the infected premolar tooth. In the second case an unusually large sequestrum developed from an infection which probably spread because of the low resistance of the patient. Bare necrotic bone was visible in the mouth for several weeks; finally, a sequestrum was separated and could be removed without causing a fracture of the jaw. The third case was that of osteomyelitis superimposed on an old fracture, with sequestration of the major part of the ascending ramus. The fourth case was very difficult to diagnose. Fulminating tissue growth was noticed in the region where a tooth had recently been extracted. Lesions which do not heal after extraction and present bone erosions are generally due to carcinoma. In the case presented, the unexpected diagnosis was tuberculosis. After the diagnosis of the mouth lesion was made, a general examination was advised, and advanced pulmonary tuberculosis was discovered.

### Case 78

#### Osteomyelitis

A. B. (468813), a 32-year-old woman, was referred to the Dental Clinic by her local doctor on Oct. 27, 1944. She complained of a swelling in the lower right premolar region of the jaw, which her doctor had treated by incision and drainage. Since then there had been continued pus discharge.

A painful swelling of the lower right jaw developed on August 30; she was given medication for pain, antiphlogistine, and ice packs. She had fever but no chills. Three days later an abscess ruptured inside the mouth with relief of the pain, but the swelling persisted. One week after onset, the patient went to a hospital where incision and drainage were performed under a general anesthetic, and a drain was inserted. Granulation tissue formed in the sinus, which the doctor had cauterized. She also had been given supportive treatment.

Examination showed a poorly nourished white woman with a draining sinus below the right mandible, midway between the chin and the angle of the jaw. The patient wore an upper plate, all maxillary teeth having been extracted ten years before. The lower teeth showed extensive dental caries. There were small submaxillary and cervical nodes on the right. The patient had lost 15 pounds. Her temperature was 99.6° F., and her pulse was 90.

X-ray examination showed several carious teeth, and some resorption of the root apex of the left lower first premolar which was badly decayed. There was marked alveolar resorption about the lower incisors. A sequestrum was present at the lower border of the mandible on the right (Fig. 359).

On Nov. 6, 1944, the patient was admitted to the House. She was given penicillin, 12,000 units intramuscularly every three hours, and vitamins and

iron. A sequestrectomy was performed on November 7 under gas-oxygen-ether anesthesia. The scar to the left of the chin was excised down to the bone. The periosteum was incised, and the sequestrum located and removed. The sequestrum was about  $1\frac{1}{2}$  inches in length and consisted of the cortex of the lower border of the mandible. The remaining bone appeared normal and no further procedure was necessary. Sulfanilamide powder was dusted into the wound, after which the subcutaneous tissue was closed with catgut, and the skin with subcuticular sutures. The infected premolar was extracted.



Fig. 359.—X-ray showing carious first premolar with periapical infection and osteomyelitis with sequestration at the inferior border of the mandible.

On November 9 the white blood count was 10,900, hemoglobin 85 per cent Tallquist, and the Hinton test negative. Pathologic examination showed a shell of bone measuring 2.2 cm. and 1 cm. in diameter, and soft red tissue measuring 8 mm. in diameter. Diagnosis: acute and chronic inflammation.

The patient received 96,000 units of penicillin intramuscularly per day for nine days; on the tenth day, 84,000 units were given. She also was given vitamins, iron, and 2 c.c. vitamin B complex intramuscularly once a day. The postoperative course was uneventful, and the patient was discharged to the Outpatient Department on November 17. She was seen again on November 22, when the wound had healed by first intention but the scar was drawn into the area where the sequestrum was removed. There was some soreness on the side of the mouth where the right lower first premolar had been extracted. The patient was advised to use warm saline mouthwashes, and was discharged to return in two months for a plastic repair of the skin defect.

**Case 79**

**Osteomyelitis and Bone Necrosis**

E. P. (457578), a 30-year-old man, somewhat debilitated by chronic alcoholism, was admitted to the hospital on July 27, 1944, with a swelling of the right submandibular area. The teeth from the first incisor to the first molar on the right were loose.

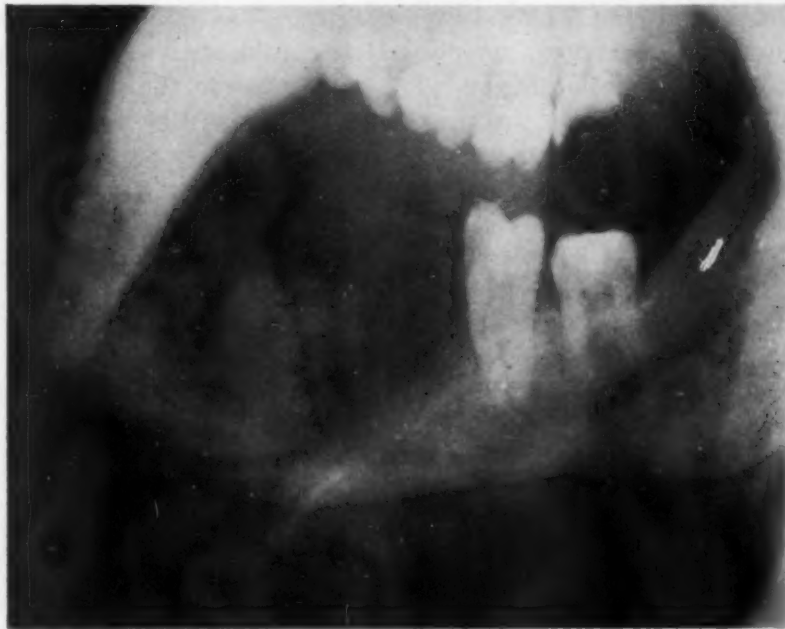


Fig. 360.—X-ray showing necrotic bone being sequestered.



Fig. 361.—Final result after sequestrectomy.

X-ray examination showed mottling of the mandible on the right, indicating osteomyelitis. The process extended approximately from the right lower incisor to the second molar.

The patient was put on sulfadiazine, 3 Gm. to start and 1 Gm. every four hours. Urine examination showed specific gravity 1.030, albumin plus, rare red blood cells, 4 white blood cells, and mucin. Hinton test was negative.

Under endotracheal gas-oxygen-ether anesthesia, and after the usual preparation of the skin and mouth, the right lower first and second incisors, canine, two premolars, and first molar were extracted. The interdental septa were removed by means of rongeur forceps. Considerable pus was evacuated, particularly from the premolar region. The outer plate of the mandible was cut away. Sulfanilamide powder was placed into the wound which was packed with a boric strip. An incision was made underneath the chin parallel with the inferior border of the jaw. The periosteum was incised and a rubber-dam drain inserted and sutured to the skin. A dry pack was applied and held in place by an elastic bandage. The patient was given an intravenous infusion of 1,500 c.c. glucose and saline.

Culture of the pus from the mandible showed abundant nonhemolytic streptococci, beta hemolytic streptococci, and *Staphylococcus aureus*. The patient received 200 mg. ascorbic acid daily and yeast tablets.

Three days after the operation the continuous sutures were removed, as there was considerable drainage of pus. One week after operation the discharge had decreased, but the white cell count was 13,200. Penicillin therapy was started on August 9, 100,000 units per day, and continued for fifteen days.

The patient was discharged on August 24, improved, to be seen at the office. A bare piece of bone was protruding through the gingival incision. This became a little loose on September 8, but since the x-ray (Fig. 360) showed it to be a major part of the jaw, it was left in place until an involucrum could form to prevent fracture of the bone. The sequestrum was finally removed on November 27, after which the wound stopped draining and healed rapidly. The patient was permanently discharged on December 4 to his dentist for the construction of a denture. An x-ray taken at this time showed a very satisfactory condition of the bone (Fig. 361).

#### Case 80

##### Osteomyelitis of Ascending Ramus

E. G. (462359), a 39-year-old man, was referred from another hospital on Sept. 20, 1944, with a diagnosis of osteomyelitis of the mandible.

Five months previously, following the extraction of two teeth, the patient had developed an acute osteomyelitis of the left mandible. This was treated at the other hospital with sulfonamides, penicillin, and incision and drainage. Several draining sinuses persisted, however.

Three years before the patient had been in a motorcycle accident and was unconscious for two days. Following this, he stated that he had been mentally confused at times, was unable to smell, and could not remember things. Four months after the accident he attempted suicide by gas. There was a history of gonorrhea eleven years before, and iritis ten years before, and allegedly he had ptosis of the kidneys.



Examination showed a swelling at the angle of the left jaw with two draining sinuses. The cervical nodes were enlarged, but no submental nodes were felt. The patient complained of pains in the jaw and head. There seemed to be a strong psychic background to much of his trouble. The present weight is 139 pounds, only 10 pounds below normal.

X-ray examination revealed that the greater part of the ascending ramus of the left lower jaw had been destroyed, and there were several large sequestra in this area. The largest measured 4 cm. in length. There was one measuring  $1\frac{1}{2}$  cm. just at the end of the jaw, and also an area of destruction extending into the horizontal ramus. Additional x-rays taken five days later showed what seemed a fracture of the base of the condyle on the left, in addition to the extensive destruction of the ascending ramus. The findings were due to an old fracture with secondary osteomyelitis (Fig. 362).



Fig. 362.—Osteomyelitis following vertical fracture of the ascending ramus.

The patient was given bed rest and oral vitamins. The culture from the draining sinuses showed *Staphylococcus aureus*, sensitive to penicillin. The white cell count was 10,700 and the hemoglobin 65 per cent.

On September 26, under gas-oxygen-ether anesthesia, and with the usual preparation of the skin of the face, a sequestrectomy was performed. An incision about 2 inches in length was made below the angle of the jaw on the left. The platysma was divided and the mandible exposed. The masseter muscle was detached so that the entire outer aspect of the jaw could be inspected. A defect filled with granulation tissue became visible and in this was found a sequestrum about 1.5 cm. in diameter. In the posterior angle another sequestrum was located, which was only partly detached. It was about  $1\frac{1}{2}$  by  $\frac{3}{4}$  inches and

included a large section of the posterior part of the ramus, extending as far as the subcondylar area. This was removed easily. The cavity was found to be completely lined by granulation, and it could not be conclusively demonstrated whether a pathologic fracture existed at the neck of the condyle. There was clear evidence, however, of an old fracture which extended diagonally through the ramus down from the mandibular notch to the inferior border about 1 inch anterior to the angle of the jaw. This fracture had healed with slight displacement of the posterior fragment. Sulfanilamide powder was placed into the wounds, a rubber-dam drain inserted, and the anterior part of the incision closed with catgut, and the skin with subcuticular sutures.

The patient received 500 c.c. of 5 per cent dextrose in water intravenously after the operation. The next day he was given penicillin, 96,000 units intramuscularly per day. This therapy was continued for seven days, together with vitamin therapy, namely, vitamin B complex, vitamin C, and A B D capsules. He was delighted with the improvement of his jaw, which he could move without pain or restraint. There was a small discharge of pus on the next three postoperative days. The wound was irrigated with saline daily.

The pathologic report of the tissue removed was sequestra from osteomyelitis with acute and chronic inflammation. There were three irregular brown carious fragments of cancellous bone covered with a gray purulent fluid, measuring 3 cm. together.

X-rays taken on October 4 showed another small sequestrum in the region of the angle of the jaw.

On October 6, under gas-oxygen-ether anesthesia, this sequestrum was located through the posterior part of the former incision, which was open and draining. This sequestrum was removed, a drain inserted, and the sutures from the anterior two-thirds of the incision removed. The right lower premolar was extracted because it was loose and sore. The patient received 1,500 c.c. of 5 per cent dextrose in water intravenously.

The area was irrigated with saline daily, and the patient was discharged to the Outpatient Department on October 8. The postoperative x-rays showing the sequestra removed are seen in Figs. 363 and 364.

When the patient was seen on November 2, there was no discharge from the wound.

On Jan. 7, 1945, the patient, who had been admitted to the psychiatric ward of the hospital on Dec. 20, 1944, because of a post-traumatic syndrome, complained of tenderness and erythema, and swelling below the left mandible. This swelling was fluctuant with a head forming. X-ray examination showed that the sequestra had been removed. There was some irregularity of the bone, but no evidence of osteomyelitis.

On January 8 the abscess opened spontaneously. Culture showed *Staphylococcus aureus*, penicillin positive. Irrigations were given every day, but one week later there was still swelling at the inferior border of the mandible with a fistula about 2 cm. below the mandibular border which continued to drain pus. Penicillin treatment, 96,000 units per day, was begun, and continued until February 3, when the fistula had healed and the swelling had disappeared. He was referred to the Eye and Ear Infirmary on February 4 for removal of his tonsils. The function of the jaw was satisfactory.



Fig. 363.—X-ray taken after sequestrectomy, lateral view.



Fig. 364.—Anteroposterior view taken after sequestrectomy.

## Case 81

**Tuberculous Osteomyelitis of the Mandible  
and Pulmonary Tuberculosis**

C. H. (463351), a 54-year-old man, presented himself on Sept. 11, 1944, at the Dental Clinic, complaining of a sore mouth. He had had a dental extraction in April which had "never healed."

Clinical examination revealed a neoplastic formation in the right lower third molar region, and a palpable node in the right submaxillary triangle. The patient was sent to the Tumor Clinic for evaluation. There it was felt that he had a new growth on the right lower alveolus about 1 inch in diameter, with cavitation in the bone and extension into both the lingual and buccal aspects. It was decided that the patient should have a biopsy examination of the tumor.

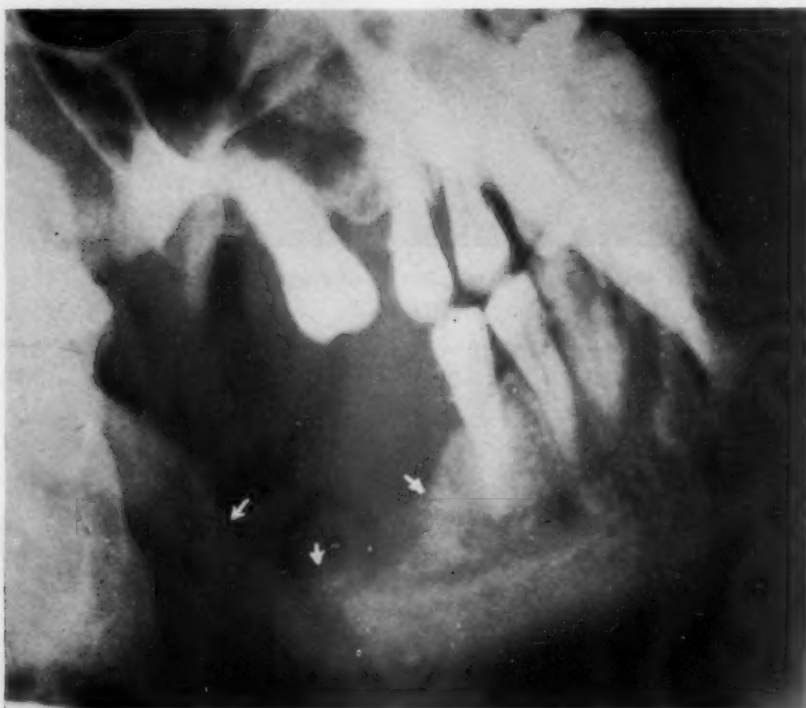


Fig. 365.—X-ray showing bone destruction due to tuberculous infection.

On September 18, the lingual and mandibular nerves were blocked with monocaine and epinephrine, and a piece of tissue was excised from the lingual aspect of the lesion. The wound was treated with the electric cautery to stop bleeding. During the biopsy procedure, the patient had an attack that resembled cardiac failure, losing consciousness. In February, 1944, he had had a syncopal attack, and another, one week previous to admission. He was referred to the Medical Clinic.

X-ray examination on September 19 showed evidence of bone resorption in the molar region of the mandible on the right (Fig. 365). There were a few small cavities in the teeth, and some marginal resorption due to periodontal disease. There was no evidence of any retained roots or apical abscesses.



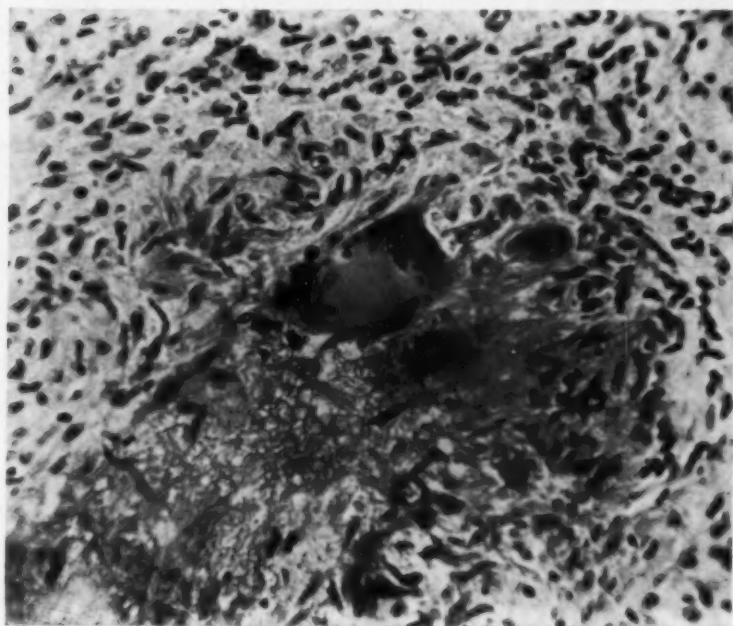


Fig. 366.—Photomicrograph showing typical tuberculous giant cell.



Fig. 367.—Chest film showing moderately advanced tuberculosis of both lobes.

The pathologic report of the biopsy showed tubercles with characteristic giant cells (Fig. 366). The diagnosis was tuberculosis of the mandible.

Examination at the Medical Clinic revealed that the patient had lost considerable weight, 25 pounds in five years. The hemoglobin was 15.2 Gm. He had no chest pain and no cough, but moderate dyspnea. The chest was clear to percussion with bronchial breathing at the left base. The heart was normal with some tachycardia but no murmurs.

A chest film on September 26 showed streaky areas of increased density and calcification extending from the hilar shadows upward into both upper lobes. Both upper lobes were considerably decreased in size. There were a few irregular areas of cavitation either due to bronchiectatic cavities or tuberculous cavities in both upper lobes. The lower lobes were emphysematous. Both hilar shadows were drawn upward. The heart was not remarkable. The findings were those of a very long-standing bilateral, moderately advanced tuberculosis of both upper lobes (Fig. 367). The question of activity could not be decided from the x-ray films.

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### ANKYLOSIS OF THE JAW

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KURT H. THOMA, D.M.D., HENRY D. HOWE, D.M.D., AND MARTIN WENIG, D.D.S.

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A patient who was admitted to the Orthopedic Department with the diagnosis of Marie-Strümpell's arthritis, and had received treatment (cup arthroplasty) for an ankylosis of the hip and two lumbar osteotomies for fusion of the spine, was seen by us because of bilateral ankylosis of the mandibular joint. A bilateral condylectomy was performed, the operative procedure being somewhat complicated because of fusion of the spine which made it necessary to perform the operation in a somewhat awkward position. Both sides were operated upon at one time with a good result. This was the second case seen by us in which ankylosis of the jaw was associated with Marie-Strümpell's disease.

#### Case 82

#### Bilateral Condylectomy

N. B. (6826), a 27-year-old man, was admitted to the hospital for the fifth time for treatment of Marie-Strümpell's rheumatoid arthritis. The patient could open his mouth only about 1 centimeter.

Examination showed that the patient had partial ankylosis of the mandibular joint. The hinge motion was 20 per cent normal; lateral and forward motion was zero. The upper teeth protruded somewhat, which might have been due to feeding habits (Fig. 368). The main disability was due to ankylosis. Osteoarthrotomy or condylectomy was advised.

In x-ray examination the left temporomandibular joint appeared to be fused (Fig. 370), and the condyle was somewhat hazy in outline. On the right side, the condyle showed practically no motion and was flattened.

On Nov. 14, 1944, a bilateral condylectomy with extraction of the left first molar was performed. Under gas-oxygen-ether anesthesia, the patient was positioned on the right shoulder so that the left side of the face would be in a horizontal plane. The head was supported by sandbags. After the usual



Fig. 368.—Photograph showing extent patient was able to open mouth.

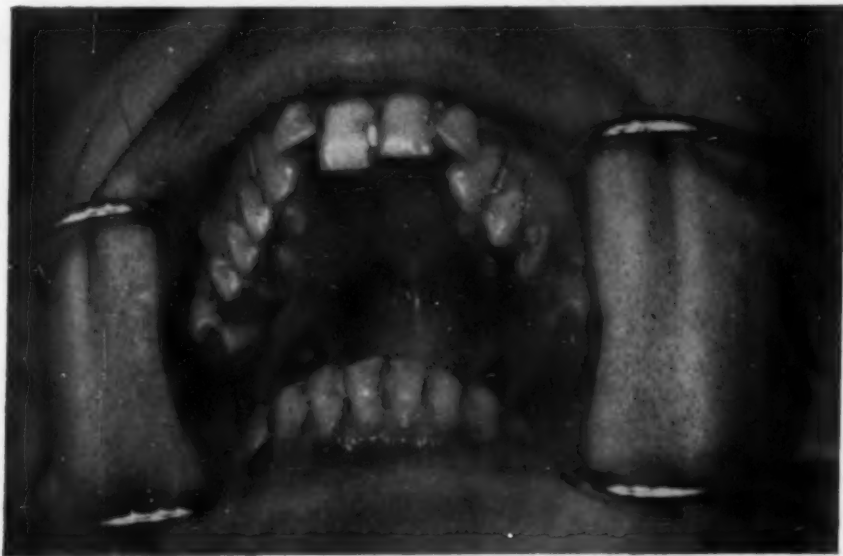


Fig. 369.—Photograph showing extent to which patient could open mouth after operation.

preparation of the temporal region and ear, 1 per cent monocaine with epinephrine was injected into the site of the incision to get hemostasis. An angulated vertical incision was made in front of the ear, extending as far as the attachment of the lobe, after which the subcutaneous tissue was divided by sharp dissection. The superficial orbital and transverse facial arteries and veins were ligated and cut and the zygomatic arch exposed. The periosteum

was incised and the bone laid bare over the area of the mandibular joint. A very slight amount of motion could be observed in the joint space, when moving the patient's jaw under the drapings. The neck of the condyle was exposed and divided by means of burrs and osteotomes. After the osteotomy was completed,

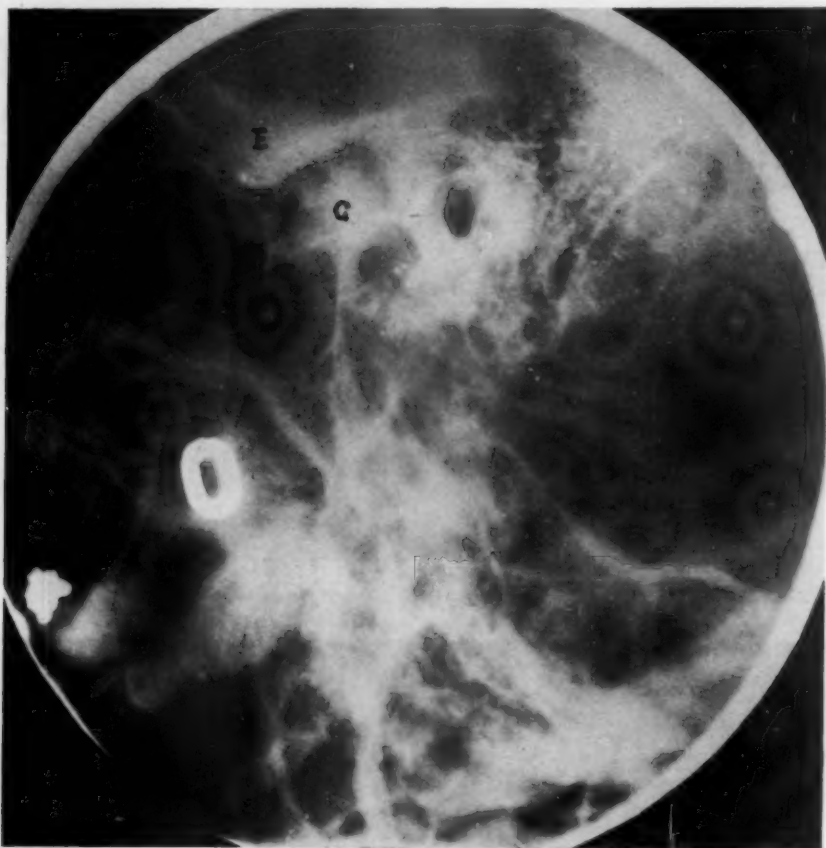


Fig. 370.—X-ray of temporomandibular joint showing ankylosis of the condyle, *C*; *E*, eminentia articularis.

an osteotome was forced into the joint space in three or four places until a cleavage plane was obtained. The condyle was then grasped with bone forceps and removed after the capsule as well as the external pterygoid muscle had been detached. Examination showed the articular surface as well as the glenoid fossa to be very much eroded and the meniscus had completely disappeared. The stump of the mandibular ramus was made smooth with back-biting forceps and bone files. After some muscular oozing was arrested by means of adrenalin packs,  $\frac{1}{2}$  Gm. sulfanilamide powder was inserted into the wound. The subcutaneous tissues were closed with interrupted catgut, and after a drain was inserted to prevent the formation of a hematoma, the incision was closed with a subcuticular suture.

With an entirely new setup, the same operation was performed on the right side, after which the mandible was tested and found to be freely movable. The right upper first molar, which was infected, was removed. The patient's





Fig. 371.—Section through excised condyle. *A*, Articulating surface; *E*, external pterygoid muscle; *C*, site of osteotomy.



Fig. 372.—Photograph of patient five months after operation.

blood pressure fell during the operation, and he was given an intravenous infusion of 1,500 c.c. of 5 per cent dextrose in water.

The next two days the patient received an intravenous infusion of 5 per cent dextrose in water plus 2.5 Gm. sulfadiazine. On the day following the operation 2 to 3 c.c. of blood had oozed from each wound. The drains were removed and the areas washed with boric alcohol, and the dressings changed. On November 18, the patient was given 72,000 units penicillin intramuscularly, and 96,000 units on each of the next three days. On November 19 there was no discharge from the incisions and only a light amount of swelling. On November 20 the function was tested. The patient could open his mouth  $2\frac{1}{2}$  cm. (Fig. 369); there was free hinge motion and good lateral motion by means of the temporal muscle, but practically no forward thrust because both external pterygoid muscles had been detached when the condylectomy was performed. There were no neurological defects. When seen three weeks later, the patient could open the mouth well and he had some lateral movement. High spots on the teeth were ground down to improve the occlusion, with a good result.

The patient was seen again in March, 1945. He had good motion of the mandible and had not lost any of the ability to open the mouth. The scar in front of the ear was not objectionable (Fig. 372).

Pathologic examination shows dense, compact bone (Fig. 371).

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## DEFORMITIES OF THE JAWS

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### A COMPARISON OF TWO METHODS OF TREATING APERTOGNATHIA

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KURT H. THOMA, D.M.D.

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Severe deformities of the jaws are generally associated with malocclusion such as apertognathia (open-bite) and mandibular protrusion or retrusion. In children, such deformities, if not excessive, can be corrected by orthodontic procedures. For adults, various types of osteotomy have been advocated.

The deformities referred to are either congenital, often inherited (Hapsburg jaw), or acquired through malunion of a fracture, through underdevelopment as in case of ankylosis of the jaw, or through excessive bone development, such as is seen if the jaw is involved by a skeletal disease such as acromegaly or leontiasis ossea.

The case to be included in this issue is one of apertognathia or open-bite; as a matter of fact, it is the most extraordinary type that has been encountered here. Besides the open-bite, the patient presented considerable prognathism. An unusual feature of this case was that a previous attempt had been made at another hospital to correct the deformity, but the attempt was a complete failure. Since the method employed at the first operation was different from the one I prefer for apertognathia, this case gives an opportunity to study the comparative value of osteotomies in the vertical ramus as against those performed in the horizontal ramus of the jaw. A review of the methods that have been devised for the correction of jaw deformities may be of interest.

According to a report by Angle (1898),<sup>1</sup> Blair was probably the first to perform an osteotomy to correct open-bite. Later, Babcock (1909)<sup>2</sup> performed a simple osteotomy, sliding the anterior part of the mandible up without tilting it. Blair,<sup>3</sup> still later, in his *Surgery and Diseases of the Mouth and Jaws*, pointed out that the jaw has to be tilted if open-bite is to be properly repaired, and, therefore, he recommended a V-shaped excision of bone just anterior to the first occluding teeth. Pickerill (1912)<sup>4</sup> recommended that the osteotomy be performed in the premolar region, while Mayerhofer (1916)<sup>5</sup> preferred the region of the first premanent molar. Korth (1921)<sup>6</sup> also pointed out that the operation should be performed in the part of the bone involved by the anomaly, namely, the horizontal ramus of the jaw. A method for the removal of a parallel section of the mandible, performed from both an intraoral and external approach, was described by New and Erich (1941).<sup>7</sup> It is used for the correction of prognathism without open-bite.

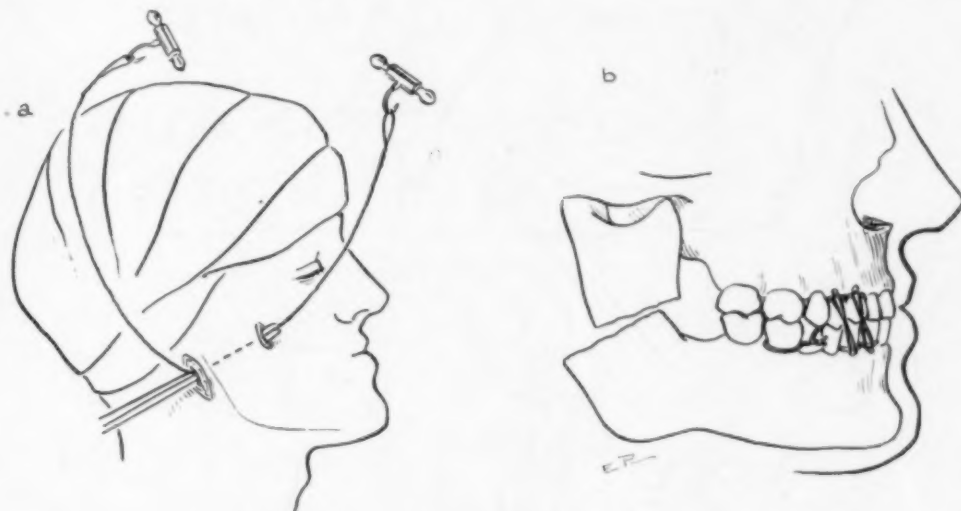


Fig. 373, a and b.—Horizontal osteotomy in ramus.

The principal methods for correcting open-bite are:

1. The horizontal osteotomy in the mandibular ramus performed with the Gigli saw (Fig. 373, a). It was recommended by Blair,<sup>3</sup> originally by Lane (1906),<sup>8</sup> and advocated by others since. Its main features are that the operation is simple, although it requires good judgment. It is claimed that it is safer than osteotomies in the horizontal ramus because infection from the oral cavity is excluded. Its advocates also claim that injury to the mandibular artery and nerve can be avoided, with the subsequent temporary numbness of the lip.

The following are the disadvantages: When the open-bite is corrected, the fragments of the rami are pulled apart at the site of the osteotomy (Fig. 373, b).

<sup>1</sup>Angle, E. H.: *Dent. Cosmos* 40: 635, 1898.

<sup>2</sup>Babcock, W. W.: *J. A. M. A.* 53: 833, 1909.

<sup>3</sup>Blair, V. P.: *Surgery and Diseases of the Mouth and Jaws*, St. Louis, 1927, The C. V. Mosby Co.

<sup>4</sup>Pickerill, H. P.: 54: 1114, 1912.

<sup>5</sup>Mayerhofer: *Ergebn. d. ges. Zahnh.* 5: No. 2, 1916.

<sup>6</sup>Korth, P.: *Vrtljschr. d. Zahnh.* 40: 272, 1924.

<sup>7</sup>New, G. B., and Erich, J. B.: *Am. J. Surg.* 53: 2, 1941.

<sup>8</sup>Lane, W. A.: *Cleft Palate and Hare Lip*, London, 1906, Medical Publishing Co.

This either results in nonunion, or the deformity recurs due to the strong action of the elevator muscles pulling the fragments back into the original position, if the intermaxillary fixation is ineffective. Direct fixation of the fragments, as recommended by Dingman,<sup>9</sup> overcomes this criticism, but it is extremely difficult to accomplish. The danger of nonunion is increased when mandibular prognathism coexists with open-bite. When the jaw is pushed back after the osteotomy has been performed, the lower fragment is medially displaced because of the angulation of the vertical ramus of the jaw. In addition, it may be stated that if the mandibular nerve is cut outside the bone, which cannot always be avoided, as the operation previously performed on my patient discloses, the numbness is quite likely to be permanent since the nerve cut before it enters the mandible does not readily unite.

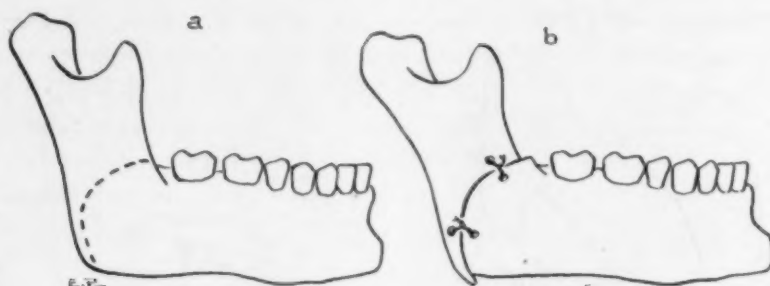


Fig. 374, a and b.—Circular osteotomy.

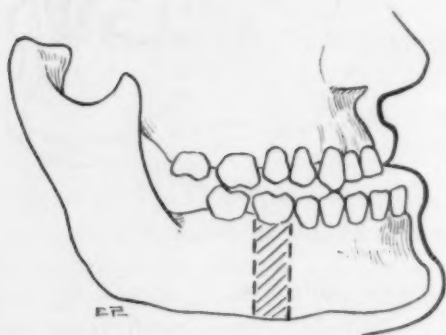


Fig. 375.

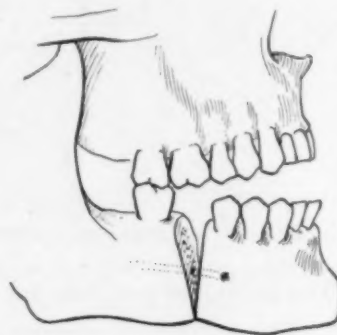


Fig. 376.

Fig. 375.—Parallel osteotomy for prognathism without open-bite.

Fig. 376.—V-shaped osteotomy for open-bite with prognathism.

2. The second method is a circular osteotomy advocated by Cryer. It is performed just anterior to the angle of the jaw, that is, behind the dental arch (Fig. 374). It allows closing the bite by rotating the anterior fragment, and is performed from an external approach. This operation is difficult to perform, and does not give much opportunity for the correction of protrusion. Kazanjian (1932)<sup>10</sup> attempted this operation by means of a trephine on one side, with a burr on the other. He found that the burr worked better.

3. In a third method, the osteotomy is performed within the dental arch, generally by sacrificing a tooth. For shortening the jaw in prognathism without

<sup>9</sup>Dingman, R. O.: *J. Oral Surg.* 2: 64, 1944.

<sup>10</sup>Kazanjian, V. H.: *INT. J. ORTHODONTIA* 18: 1224, 1932.



open-bite, a parallel piece is removed (Fig. 375). For correction of open-bite with prognathism, the V-shaped osteotomy is recommended (Fig. 376).

For the correction of open-bite without prognathism, the V-shaped osteotomy is not entirely satisfactory, because it causes the incisor teeth to be displaced too far back. A method described by me<sup>11</sup> allows closing the bite without retrusion of the mandible. I named it the "Y-shaped" osteotomy. In this method a tooth is removed with a V-shaped section of the jaw, the apex of the V reaching as far as the mandibular canal. From an external approach, the tail of the Y is cut from the inferior border of the mandible up to the canal (Fig. 377, *a* and *b*).

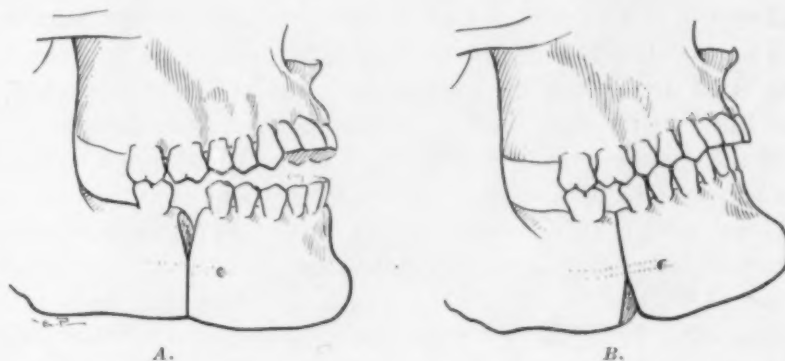


Fig. 377 A and B.—Y-shaped osteotomy for open-bite without prognathism.

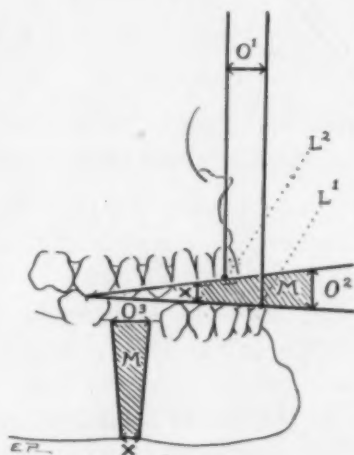


Fig. 378.—Rhomboid osteotomy for open-bite with prognathism. Diagram to determine the amount of bone to be removed to correct both prognathism and open-bite.

New and Erich's operation and the Y-shaped osteotomy can be performed entirely from an intraoral approach, if one does not aspire to the preservation of the mandibular nerve and artery. It is a debatable question, however, whether the preservation of the nerve is worth the trouble that it involves, since it is a well-known fact that the nerve, when cut within the bone, unites without much trouble in from six weeks to six months.

The method used in the case to be described was an osteotomy by which a rhomboid section of bone was excised. It was performed entirely from an intra-

<sup>11</sup>Thoma, K. H.: Surg., Gynec. & Obst. 77: 40, 1943.

oral approach. The shape of the section of bone that needed to be removed was determined by means of a geometric diagram, which is drawn with the aid of a lateral jaw film (Fig. 378). In this diagram,  $O_1$  represents the distance the lower incisor,  $L_1$ , has to be set back to come into a normal anatomical position,  $L_2$ . The distance  $O_1$  is therefore the amount of bone to be excised at the alveolar ridge,  $O_3$ . The next step is to determine the amount of tilting needed. If a V-shaped piece were removed with the opening of the V equal to  $O_3$ , the jaw would be either tilted too much, and as a result the molar teeth would not come into occlusion, or the gap  $O_3$  would not be completely closed, and, therefore, the protrusion would not be entirely corrected. It is necessary to remove a rhomboid section. The side of this rhomboid at the inferior border is anywhere from zero, which tilts the jaw the maximum amount, to  $O_3$ , when no tilting takes place. It is determined by drawing the angle of the open-bite into the diagram. One line extends from the first occlusal contact point through  $L_1$  the cutting edge of the mandibular central incisor, and another through  $L_2$  the theoretical anatomical position of the lower incisor behind the maxillary one when the teeth are closed.  $O_1$  is transferred between these lines, and is represented by  $O_2$ . The vertical dimension of the jaw is now measured and transmitted to find X, which completes the rhomboid (M), which may be cut from a heavy piece of tinfoil and used as a pattern when performing the operation. Of course there is no reason why the operation cannot be performed from both intraoral and extraoral approach as well, if one desires to preserve the nerve and prevent temporary numbness of the lip.

### Case 83

#### Apertognathia and Prognathism Corrected by Bilateral Rhomboid Osteotomy

G. H. (471435), a 25-year-old man, was referred to me for a bilateral osteotomy of the mandible to correct apertognathia and mandibular prognathism. He entered the hospital on Nov. 24, 1944.

The patient was born with a harelip and partial cleft palate. It involved only the alveolar process on the left and did not extend into the hard or soft palate. He has had a protruding jaw since he was a baby. The harelip and alveolar cleft were repaired at the age of 2 months, and another operation was done at 12 years of age. The jaw became more prominent, however, and the bite opened in the incisor region. The patient's health had been good, except for streptococcal pneumonia at the ages of 1, 10, 13, and 18 years. He was underweight, but had not lost weight recently.

One year ago the patient had an osteotomy performed on the mandibular rami at another hospital. Wires were inserted into the jaw in the premolar region to strengthen and support intermaxillary fixation. During the two months of immobilization, infection of the left jaw at the side of the wire caused a complication. The end result was unsatisfactory; it did not improve the open-bite and mandibular protrusion.

Examination showed typical facies associated with mandibular protrusion (Fig. 379). He presented an open-bite (Fig. 380) of about 1 inch and reversed



Fig. 379.—Patient with prognathism and open-bite.

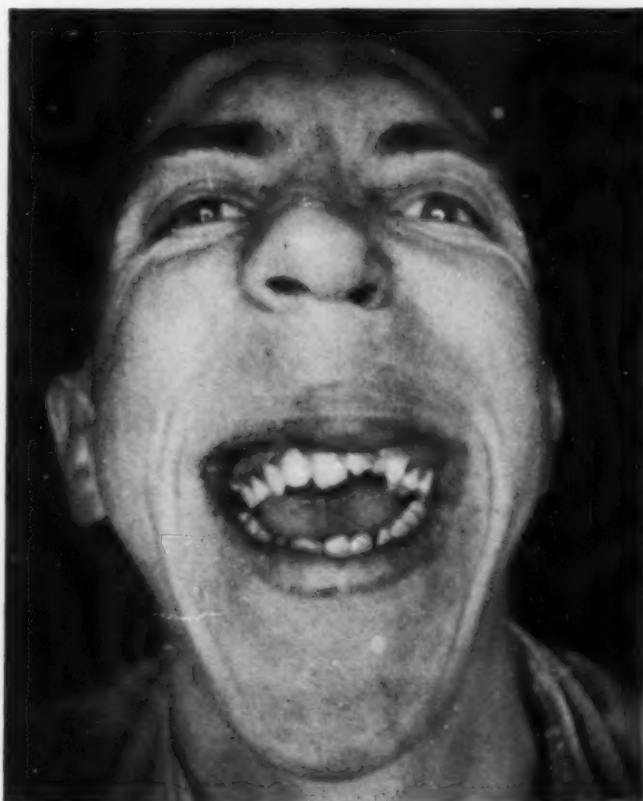


Fig. 380.—Open-bite when molars are occluded.

overbite (Fig. 382), and irregularity of the teeth in the left anterior part of the maxilla due to the cleft. He also presented a very high conical palate and a long upper lip. A perforation in the alveolar process in the labial sulcus was



Fig. 381.—Splints attached, showing patient's occlusion.



Fig. 382.—Photograph showing open-bite and prognathism in lateral view.

found on the left side of the maxilla. Neurological examination revealed an area with loss of sensation (touch and pain) at the angle of the mouth and lip on the right. He has a speech disorder related to the congenital defect. Splints had



been attached to his teeth by his dentist for fixation of the osteotomy, and immobilization of the jaw (Fig. 381). The extraction wounds of the two mandibular molars, which had been removed two months ago to facilitate the operation, had completely healed. A patch of yellowish brown skin pigmentation was seen near the left pectoral region.

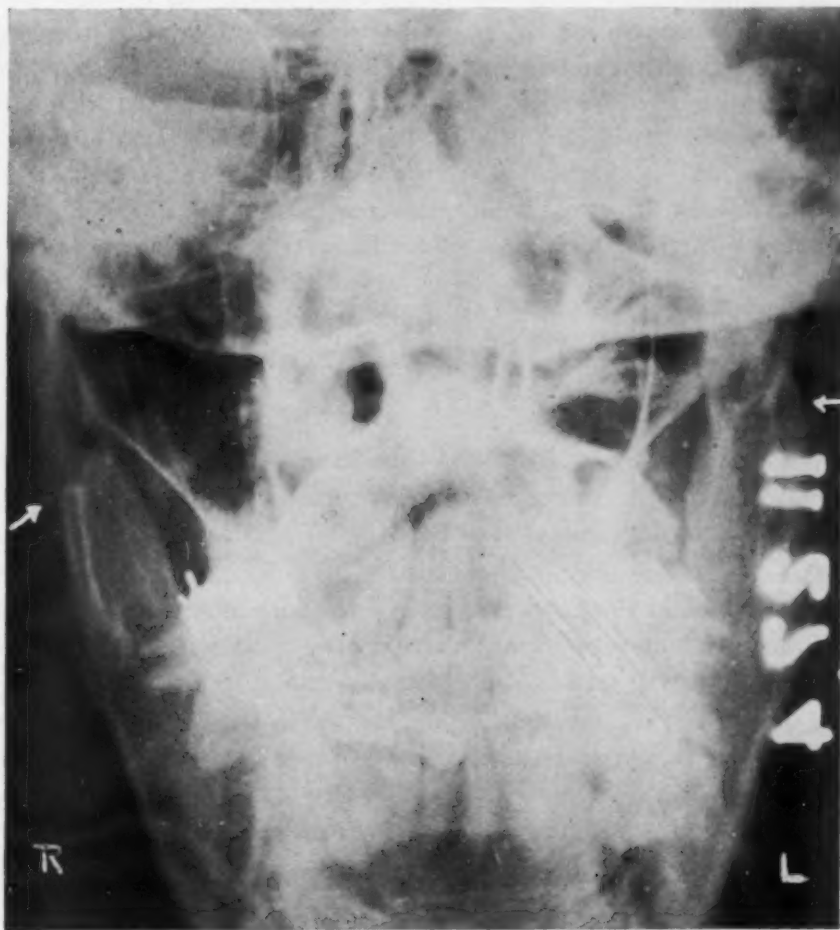


Fig. 383.—X-ray showing site of osteotomy in the ascending ramus performed one year previously at another hospital.

X-ray examination of the jaws revealed in anteroposterior views a defect in both ascending rami. These defects no doubt were caused by the osteotomy performed there; on the right a displacement which so often occurs after this operation was quite evident (Fig. 383). The lateral jaw films (Figs. 384 and 385) showed a very deep mandible; all the teeth were vital; the first permanent molars had been removed and splints attached to the teeth. X-ray examination of the chest was negative.

Blood examination showed a hemoglobin of 14 Gm.; red cell count, 4,150,000; white cell count, 8,500; bleeding time, 4 minutes; clotting time, first tube, 8 minutes, second, third, fourth, and fifth tubes, 10 minutes; clot retraction, normal; Hinton test, negative.

On Nov. 25, 1944, under intratracheal gas, oxygen, and ether anesthesia, with morphine,  $\frac{1}{6}$  grain, and atropine,  $\frac{1}{100}$  grain, as premedication, a bilateral rhomboid osteotomy for protrusion of the mandible and open-bite was performed. The skin, and mucosa of the mouth were prepared with zephiran. Four cubic centimeters of monocaine and epinephrine were injected on the outer and inner surfaces of the mandible on the right at the site of the operation to control hemorrhage. An incision was made in the alveolar mucosa on the outer surface of the jaw distal to the second premolar and extending downward vertically to the inferior border of the mandible. A second incision was made from the mesial side of the second molar, extending posteriorly and downward. A flap was prepared by detaching the mucoperiosteum from the bone. The outer surface of the mandible then came into view, and was exposed as far as the inferior border of the jaw. After protecting retractors were carefully placed, a circular saw was used to make two vertical cuts in the bone. The distance between the two cuts had been determined and was equal to the width of the space created by the extraction of the first molar at the alveolar crest. They converged toward the inferior border, according to a predetermined pattern. The alveolar nerve and artery were cut. Only slight hemorrhage was produced, and this was arrested by means of adrenalin packs. Since the saw did not reach the lingual part of the inferior border, burrs, and chisel were used to complete the excision of the segment of bone. After its removal, a wet sponge was inserted into the wound.

The other side of the jaw was then injected with monocaine and epinephrine on the inner and outer surfaces, and the same operation was then performed on the left side of the mandible. This freed the anterior segment completely.

The jaw was then positioned by pushing it back into contact with the posterior fragment, and tilted up, so that the mandibular incisors extended up and behind the maxillary incisors. A threaded piece of wire was inserted on each side of the previously prepared and attached splint, and, by means of a nut inserted on each side, the fragments of the jaw were approximated and fixed on both sides. When the mandible was occluded, it was found that the first molar in the upper jaw on both sides was elongated, and it had to be ground down somewhat, especially on the lingual part of the occlusal surface. This brought about as good occlusion as could be expected with the irregularity of the incisor teeth. The incisions were then closed by means of silk sutures, and the upper and lower splints connected with stainless steel wires to hold the teeth in the new occlusal position and immobilize the osteotomized mandible (Fig. 388). Great improvement was at once noted in the patient's appearance and shape of the mouth (Fig. 389). The mandibular protrusion had disappeared, and the lips, which formerly could not be easily brought together, came easily into contact.

The patient received an intravenous infusion of 2,500 c.c. of 5 per cent glucose during the operation, and the same amount the following day. He did not lose much blood, and after a Barton bandage was applied, he was discharged to the ward in good condition. Sulfadiazine was given postoperatively because of the bone infection which he was supposed to have had after the

previous operation. The patient's mouth was irrigated daily, and later sprayed with an antiseptic solution (Cepacol).

Postoperative x-rays in anteroposterior and lateral views were made. They showed the old osteotomy in the vertical rami previously mentioned, and the recent osteotomy on both sides of the mandible in the region of the removed

Fig. 384.



Fig. 385.



Fig. 386.



Fig. 387.

Figs. 384 and 385.—X-rays showing the site of the osteotomy, the first molars having been extracted previously.

Figs. 386 and 387.—Postoperative x-rays after a rhomboid osteotomy.

first molars (Figs. 386 and 387). Comparison with films taken previously showed that the marked malocclusion has been corrected. The fragments are in good position and held by intermaxillary fixation with splints wired to the teeth.

Recovery was uneventful. On December 1 the sulfadiazine level was 7 mg. per cent, and chemotherapy was discontinued on December 3. The patient

received a high protein liquid diet with vitamins containing sufficient calories to maintain his weight. He was discharged on Dec. 10, 1944, and seen at my office for spraying and supervision. After six weeks the intermaxillary ligatures were removed and the healing tested. The right side was quite firm, but on the left, where unnoticed the bands of the splint had become loose, the union was

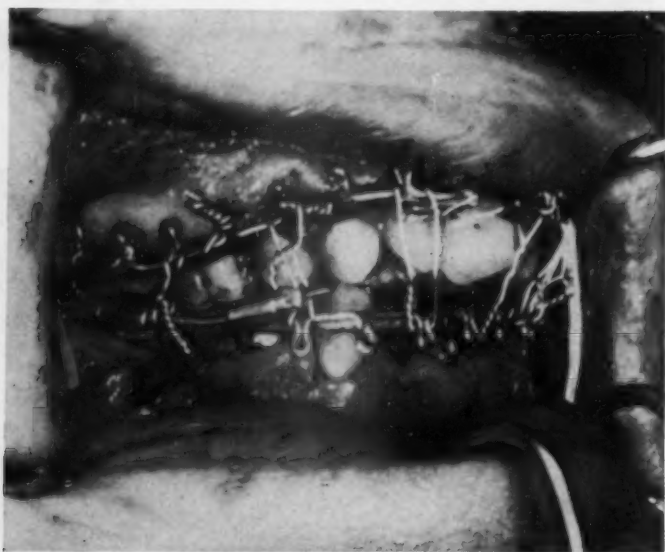


Fig. 388.—Teeth wired into occlusion after the osteotomy had been performed.



Fig. 389.—Occlusion of lips is improved after operation.

quite weak. The bands were reinforced by wiring and the mandible immobilized for two more weeks. At the end of the eighth week, however, both sides were firm, and the patient was allowed to masticate food with the support of the arch wire remaining in the lower jaw. After the ninth week the remainder of the appliance was removed; the patient was given a prophylactic treatment and discharged, relieved of his deformity, to the care of his local dentist to have the anterior teeth in the upper jaw improved by means of a bridge. The patient was greatly pleased with the change in his appearance (Fig. 390), and with his increased ability to chew food.

*Discussion.*—This case illustrates the superiority of the osteotomy in the horizontal ramus over that of the ascending ramus. In osteotomies in the



latter, the difficulties arise from the fact that it is not easy to get the right direction and the right level when cutting the rami, almost blindly, with a Gigli saw. The disalignment in which the fragments healed in this case, as shown in the x-ray (Fig. 383), and the permanent anesthesia (one-year duration) in the lip which resulted, although it is supposed to be avoided by this operation, are evidence of this contention. The reason that the open-bite as well as the protrusion recurred may have been a distraction of the fragments after the open-bite was reduced, followed by a gradual drawing together by muscle spasm, against insecure intermaxillary fixation.



Fig. 390.

Fig. 390.—Postoperative photograph.



Fig. 391.

Fig. 391.—Postoperative photograph showing occlusion.

In the rhomboid osteotomy, on the other hand, there is very little muscle pull to displace the newly positioned fragments, and the resection of a piece of bone, the size of which has been correctly predetermined, makes the correction of the deformity an accurate procedure. The only complication to be feared is infection, but this danger today can be greatly decreased by careful sterilization of the mouth, the use of a strictly aseptic technique, the prophylactic therapy with sulfonamides or penicillin, and the building up of the patient's resistance by prescribing a proper diet and by promptly administering blood transfusions if the patient has lost a great deal of blood or is anemic or below par for any reason.

## TUMORS OF THE MOUTH AND JAWS

KURT H. THOMA, D.M.D., HENRY D. HOWE, D.M.D., AND  
MARTIN WENIG, D.D.S.

A number of interesting cysts and other tumorous new growths were seen in the clinic. We present a pregnancy tumor case with a history of epilepsy controlled by the use of phenobarbital for a period of eight years. The diagnostic problem was to decide whether this condition was due to a hormonal disturbance occurring during pregnancy, or whether phenobarbital may, in certain patients, cause similar gingival hypertrophies as are commonly seen in epileptics who receive dilantin sodium medication. The pathologic examination showed it to be a pregnancy tumor.

A patient is presented whose mandible was greatly enlarged. On roentgen examination, it showed osteolytic areas such as those seen in osteomyelitis, but which, after biopsy examination, were found to be due to Paget's disease. This is a case of Paget's disease of the mandible without involvement of any other bones.

A large cyst is also included; it was thought to be an adamantoblastoma because of its extent and the way it spread under the incisor teeth of the mandible. It is of interest because the incisor teeth which were saved remained vital. Another cystic lesion with an x-ray appearance characteristic of adamantoblastoma, and which had been operated on twice before with a diagnosis of adamantoblastoma, turned out to be an ossifying fibroma.

A mixed tumor of the cheek which recurred after excision under the zygomatic arch is included, and also a tumor of the palate. The biopsy taken from the hard palate was diagnosed as a mixed tumor, but examination of the tissue from the soft palate proved it to be an adenocarcinoma. Mixed tumors may also occur in the jaws; we recall one which occurred in the maxilla\* and involved the entire bone on one side, and another in the mandible.†

Many epidermoid carcinomas are seen in the Tumor Clinic of this Hospital. A semiprivate patient presented an unusual type of lesion on the alveolar ridge of the mandible which resembled an ulceration. Another semiprivate patient with a very large tumor, such as is not often seen these days, is presented by the courtesy of Dr. Ira T. Nathanson.

Dr. David Weisberger of our clinic is presenting a patient with a swelling of the parotid gland. It is one of a long series of swellings and tumors investigated by Dr. Weisberger in an attempt to correlate clinical findings, sialography, and pathologic diagnosis.

### Case 84

#### Multiple Pregnancy Tumors

G. W. (470891), a 28-year-old woman, was admitted to the hospital on Nov. 17, 1944, for the removal of multiple pregnancy tumors.

\*Thoma, K. H.: *Clinical Pathology of the Jaws*, Springfield, Ill., 1934, Charles C Thomas.

†Thoma, K. H.: *Oral Pathology*, ed. 2, St. Louis, 1944, The C. V. Mosby Co., p. 1041.

The patient stated that her gingivae had always been spongy. When she was about two months pregnant, they started to enlarge. At night she bit into them, causing a great deal of bleeding and soreness. The condition did not regress after parturition, which was two months ago.



Fig. 392.—Multiple pregnancy tumors in upper and lower jaws.

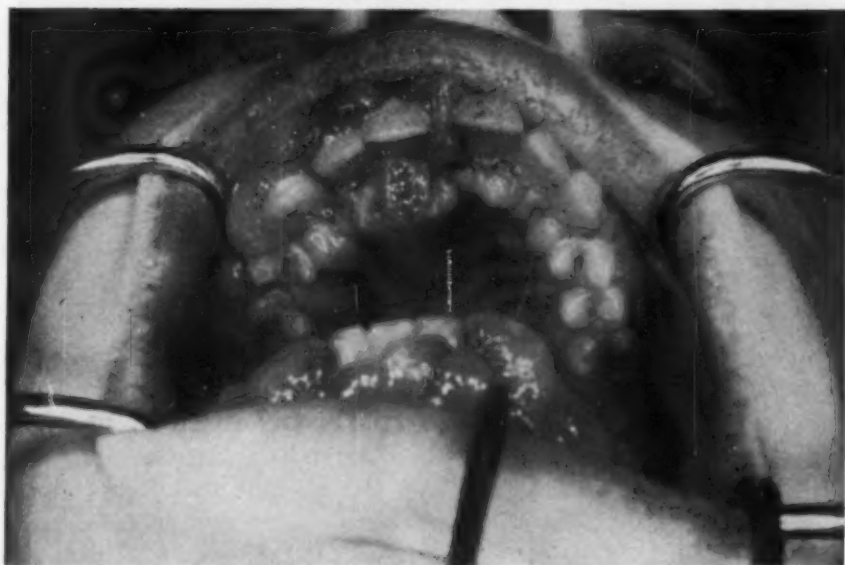


Fig. 393.—Multiple pregnancy tumors; appearance of palatal gingiva.

The patient was an epileptic and has been under the care of a neurologist. She had been taking  $1\frac{1}{2}$  grains phenobarbital for eight years. Recently the dose was increased to 3 grains per day. She has never taken dilantin sodium.

Examination of the mouth showed very marked hypertrophy of the gingival margins in both the upper and lower jaws. From the interdental papillae, spherical tumor masses extended away from the surface in many places. These were especially marked in the anterior part of the maxilla, both on the labial and palatal sides of the dental arch, and in the anterior region and the right side of the alveolar process of the mandible (Figs. 392 and 393).

X-ray examination showed some resorption of the alveolar margin, especially in the region of the mandibular molars and the maxillary incisors, but otherwise nothing remarkable was disclosed (Fig. 394).



Fig. 394.—X-ray showing bone resorption in molar region and spacing of teeth.

The patient was given sulfadiazine therapy on entering the hospital. The bleeding time was established; it was less than 5 minutes, and the clotting time 8 minutes. Her blood was typed so that in case of excessive bleeding she could be given a transfusion without delay.

On November 18, under intravenous pentothal sodium anesthesia, with an endotracheal tube inserted through the nose, and the pharynx completely packed, the mouth was prepared in the usual manner with zephiran. With the electrocautery, the tumor masses, which extended from the gingival margin both on the inner and outer aspects of the maxilla and mandible, were excised. The ensuing hemorrhage was arrested by electrocoagulation and adrenalin packs (Fig. 395). The lower left third molar was extracted, as it was loose and completely submerged in the tumor tissue. Zinc oxide and tannic acid powder and essential oils were mixed into a semisolid paste, which was applied to the teeth on the inner and outer surfaces in both the upper and lower jaws to protect the raw surfaces resulting from the gingivectomy (Fig. 396).

Pathologic report: The specimen consisted of several pieces of firm tissue, measuring 2.5 cm. all together. Microscopic examination showed a dense plasma-



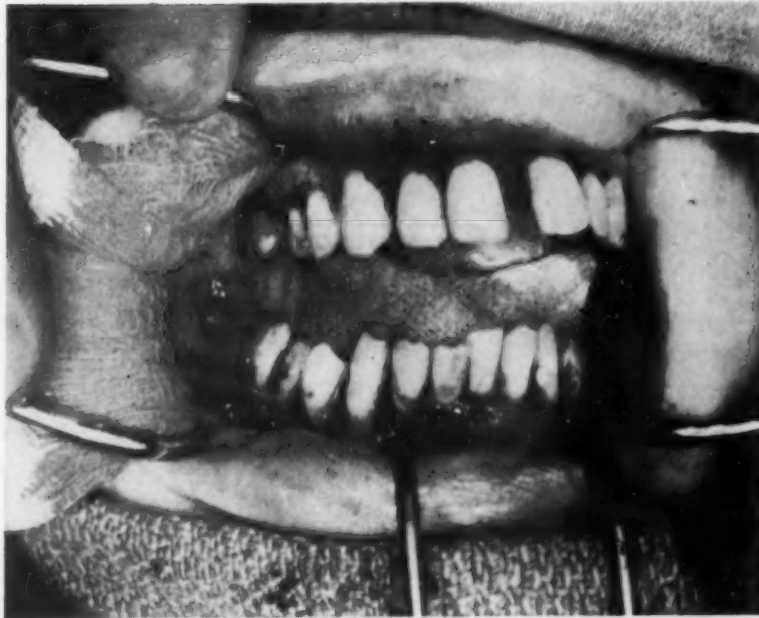


Fig. 395.—Photograph taken after performing gingivectomy.



Fig. 396.—Zinc oxide pack applied to the teeth.

cell infiltration (Fig. 398), through which were numerous vascular spaces containing red blood cells (Fig. 397). Occasional lymphocytes were also present.

Diagnosis: Pregnancy tumor.

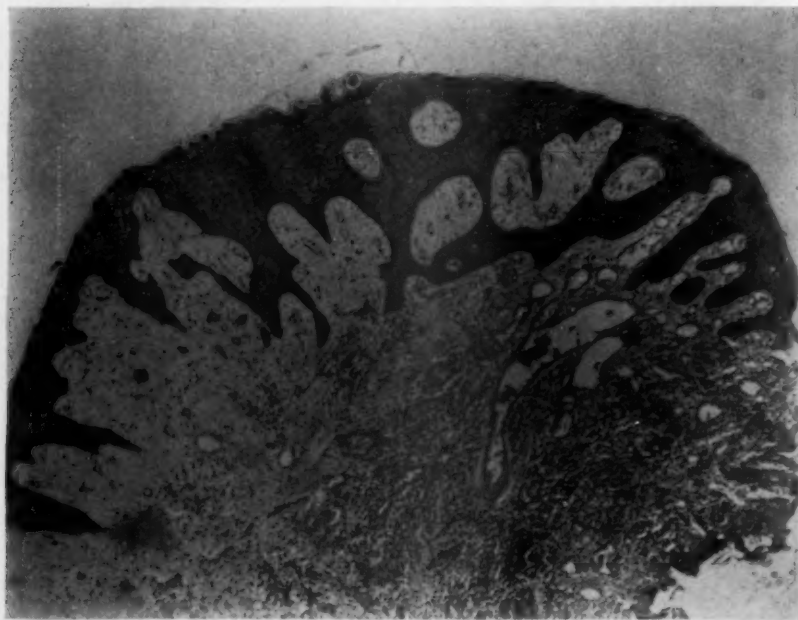


Fig. 397.—Photomicrograph of section through pregnancy tumor showing blood vessels.

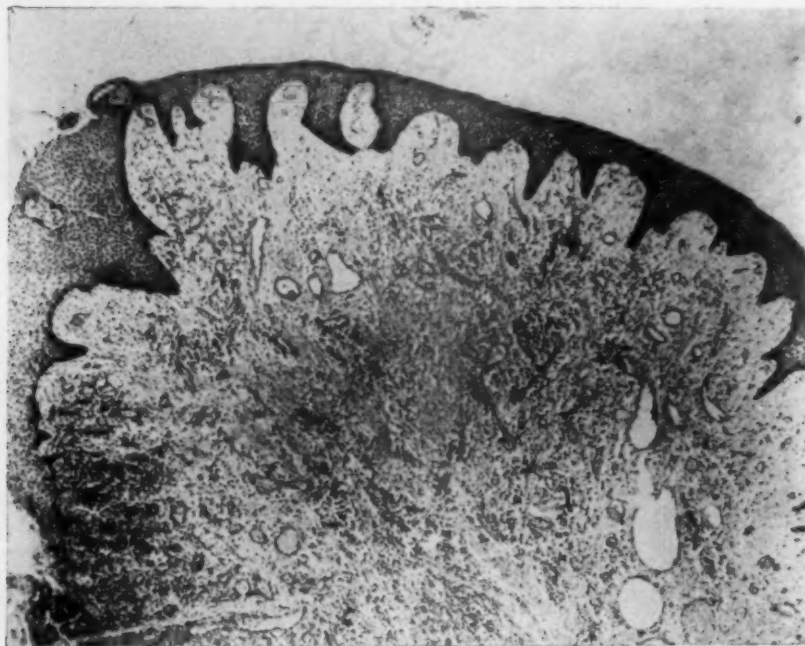


Fig. 398.—Photomicrograph of section through pregnancy tumor.

The patient complained of some soreness and sensitiveness of the teeth after the paste had been removed, but otherwise recovery was uneventful, and she was discharged on Nov. 27, 1944.

Later she complained of a tired feeling. Blood examination showed hemoglobin 10.3; white cell count, 7,400; polymorphonuclear leucocytes, 60 per cent; small lymphocytes, 34 per cent; monocytes, 4 per cent; eosinophiles, 2 per cent. There was considerable achromia, and moderate variation in the size of the red blood cells. Platelets were normal. She was referred to a physician for treatment of her anemia. Her tumors did not recur; a photograph taken in January, 1945, showed the postoperative results (Fig. 399).



Fig. 399.—Photograph taken two months after operation.

#### Case 85

##### Paget's Disease of the Mandible

A. F. (457125), a 46-year-old man, was admitted to the House on July 24, 1944, complaining of enlargement of the mandible.

X-ray examination revealed a peculiar mottling of the mandible with many osteolytic areas. A retained third molar was also seen (Fig. 400). The findings might be due to osteomyelitis. Paget's disease, which was also considered as a diagnosis, was ruled out because no other bones showed any changes, and because in instances where Paget's disease involves the skull the mandible is very rarely affected.

On July 25, 1944, under gas-oxygen-ether anesthesia, an incision was made on the alveolar crest of the edentulous mandible, extending from the left third molar region to the right premolar region. The mucoperiosteum was dissected away from the bone and retracted. The jawbone appeared normal on the surface, except in the left third molar region where there was some soft tissue. The bone was cut away and the unerupted third molar exposed and removed by elevator technique. A culture was taken. Two pieces of bone were removed in the anterior part of the mandible for biopsy examination.

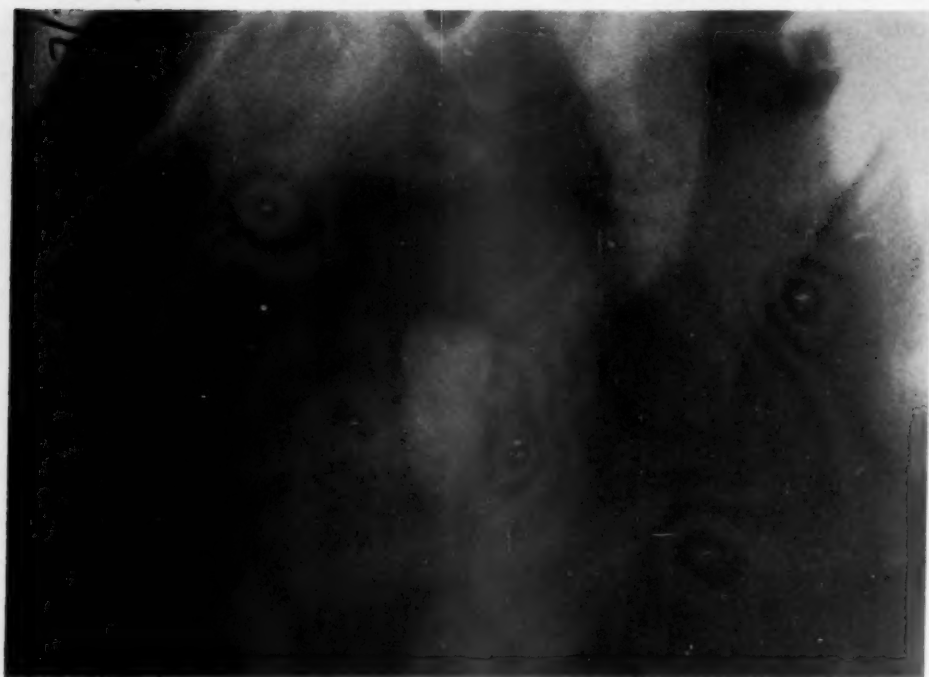


Fig. 400.—X-ray showing Paget's disease of the mandible and retained third molar.

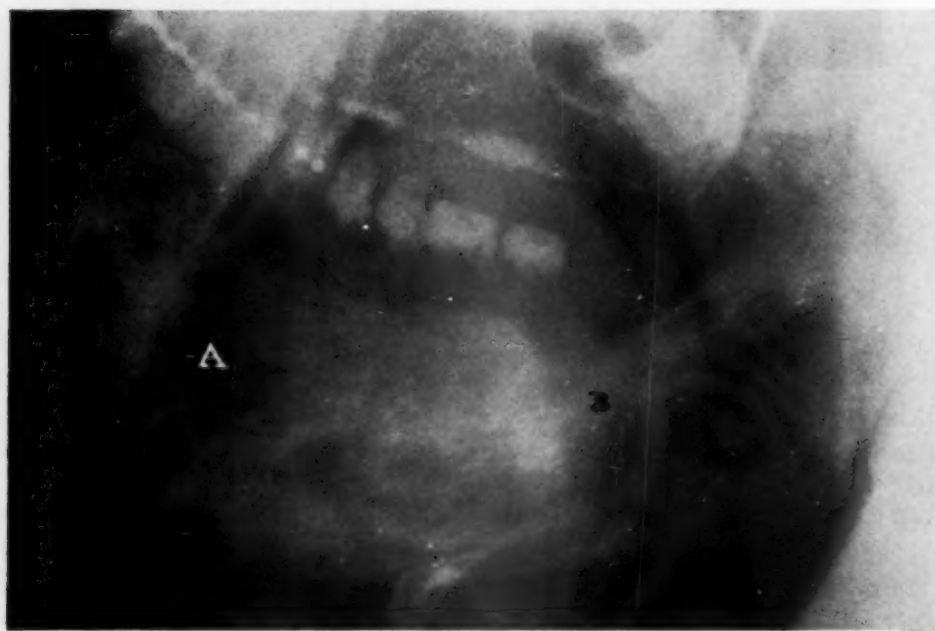


Fig. 401.—Postoperative x-rays showing site of biopsy, A, and extraction wound, B.



The bone was smoothed and the mucosa replaced. The incision was closed with continuous silk sutures after inserting sulfanilamide powder.

Postoperative x-rays showed the tooth removed and the site of the biopsy; otherwise the condition was the same as previously reported (Fig. 401).

The culture from the tooth socket showed a few nonhemolytic streptococci, *Staphylococcus aureus*, and alpha hemolytic streptococci. Sections of the bone from the mandible showed Paget's disease (Fig. 402).

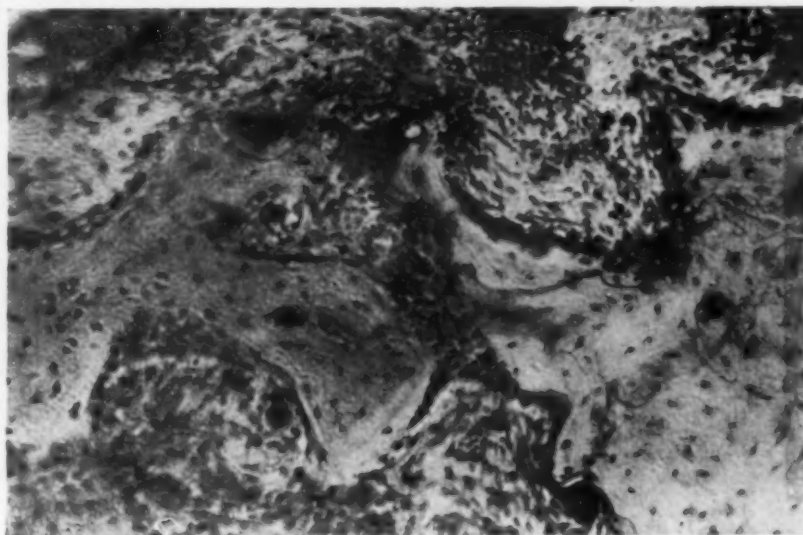


Fig. 402 —Paget's disease. Biopsy from mandible.

### Case 86

#### Dentigerous Cyst

R. R. (467445), a 21-year-old girl, was admitted to the Hospital on Oct. 17, 1944, with a cyst of the mandible.

The patient has noticed a swelling of the gingivae of the lower left jaw for the past four years. When this swelling was squeezed, pus exuded from a small opening below the lower right second incisor. There was occasional pain when the swelling was filled with pus, but this was relieved by pressing it out. Two years ago the lower left second molar was extracted. A short time after this, pus began to drain from the socket of this tooth.

X-ray examination revealed a large trabeculated cystic area in the mandible, thinning out the cortex of the inferior border of the jaw. It extended from the left third molar below the apices of the teeth to the first molar roots on the right. A deformed tooth was seen in the right canine region in this cyst (Figs. 403 and 404).

Diagnosis: Dentigerous cyst or adamantoblastoma.

The patient was given sulfadiazine, and on October 18 excision of the cyst and unerupted tooth and extraction of four teeth were performed under endotracheal gas-oxygen-ether anesthesia.

In order to excise this very extensive cyst which occupied the entire mandible, three incisions were made. The first incision was made in the anterior part

of the mandible, the labial sulcus, extending from premolar to premolar. The mucoperiosteum was detached and a large window cut into the bone. The unerupted tooth from which the cyst developed was immediately encountered.



Fig. 403.—X-ray showing extensive dentigerous cyst on the left; T, tooth.



Fig. 404.—X-ray showing extensive dentigerous cyst on the right.

The cyst membrane contained pus and cholesterin crystals, which escaped through a puncture and was cultured. The cyst sac could easily be detached from the bone, and was cut apart in the median line.

An incision was next made on the right side of the mandible opposite the premolar and molar teeth. The two premolars were extracted, and the outer alveolar plate removed to obtain a good approach to the cyst cavity here. The



Fig. 405.—Section through excised cyst membrane.



Fig. 406.—Postoperative appearance of mouth; the incisor teeth have remained vital.

cyst sac was detached from the bone and pushed into the anterior compartment, from which it was removed. Sulfanilamide powder was inserted and a borie strip.

A longer incision was made on the left side of the mandible, extending from the canine to the third molar. The mucoperiosteum was retracted on the

outside of the jaw, the second premolar and second molar extracted, and a large window cut, exposing the cyst sac here. This was detached from the bone, and was at least three times as large as the part on the right side. The sac was removed together with the anterior part which, as stated, had been cut in the median line. Sulfanilamide powder was inserted into the cavity and into the anterior part of the wound. A boric strip was placed in the posterior compartment, and the anterior part was closed with silk sutures. The cyst sac with the unerupted tooth in position was sent to the laboratory.

The patient had lost considerable blood during the operation and was given an intravenous infusion of 2,500 c.c. of 5 per cent dextrose and 250 c.c. blood plasma.

The culture from the cyst showed *Staphylococcus albus* and alpha hemolytic streptococci.

Pathologic examination: The specimen consisted of two soft irregular pieces of tissue measuring 6 by 2 by 1 cm. and 3 by 1 by 1 cm.; one piece contained a tooth.

Microscopic examination showed a cyst sac lined with stratified squamous epithelium, and its wall infiltrated with a chronic inflammatory exudate composed of lymphocytes, monocytes, and eosinophiles (Fig. 405).

The patient's mouth was washed with warm saline solution after every meals. The dressings were removed on October 21, and the patient was discharged the next day. She was given a syringe to irrigate the healing cavities. She was last seen on January 20, when the cyst on the right had completely healed, the anterior teeth appeared normal and vital by clinical and x-ray examination, and the cyst cavity on the left was gradually becoming obliterated. Saving the anterior teeth in this case proved a successful venture (Fig. 406).

#### Case 87

##### Ossifying Fibroma of Mandible

D. R., (474125), a 41-year-old woman, entered the House on Dec. 19, 1944, because of tumor of the mandible.

Twenty-six years before, the patient had had two operations for intraoral excision of a tumor, which she was told was an adamantinoma. Twelve years ago she had had a recurrence of the tumor of considerable size. This was removed from an external approach, leaving all the teeth in place. Two years later the left lower second premolar became sore, and was extracted. Since then she has had no check-up, but has had pain and swelling off and on. Two months ago the pain became severe and she has been taking injections of morphine every night. Two weeks ago the left side of the face became markedly swollen. Neurological examination revealed that there was numbness of the left cheek and lip.

The patient stated that her jaw felt like a sponge. The teeth were sore, and she was suffering a great deal of pain on the left and right sides of the face, extending from the jaw over the head.

Examination revealed asymmetry of the face due to expansion of the left side of the mandible. The skin over the left face showed loss of pain sense.



There were two scars on the neck on the left. One scar was due to an excision of a cervical lymph node done in 1918. The second scar was on the sub-mandibular area, 2 inches in length. There was no lymphadenopathy.

Intraoral examination showed that all the teeth were present except the left lower second premolar and first molar. The jaw was somewhat expanded in the anterior region on the labial and buccal sides, and was quite tender in the molar region and on the outer surfaces (Fig. 408).

X-ray examination revealed a large radiolucent area in the jaw of a cystic nature, which extended from the left lower second molar around to the second premolar on the right. The picture of the chin showed a multicystic condition involving the bone of the entire mental region (Fig. 407).

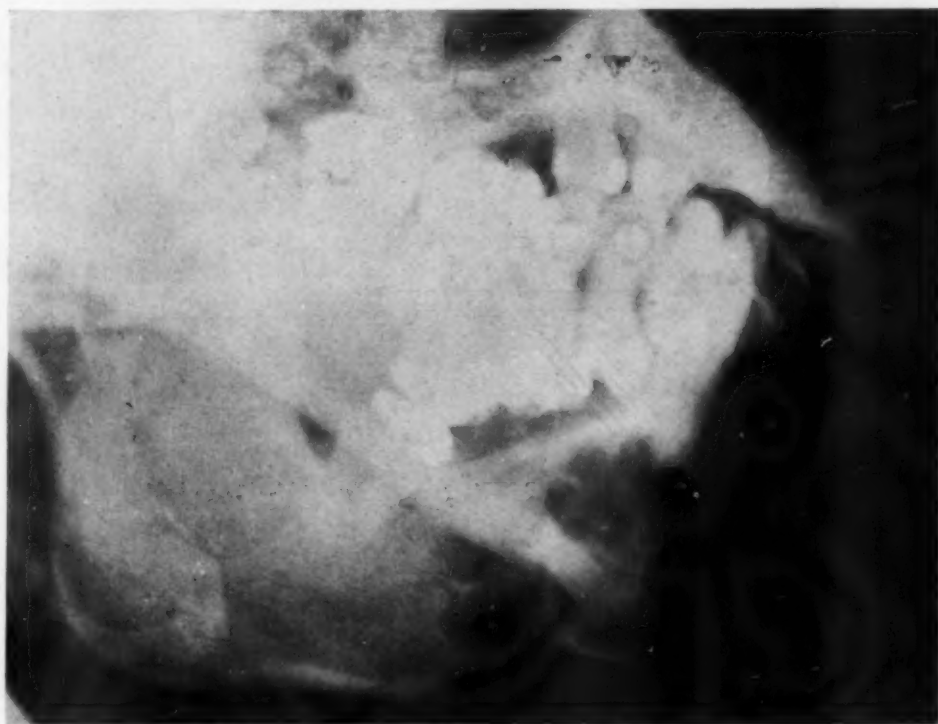


Fig. 407.—X-ray showing polycystic tumor of the mandible.

**Diagnosis:** Because of the previous findings, a diagnosis of adamantoblastoma was made, and radical excision was advised.

Blood examination showed white cell count 8,000; hemoglobin, 12.8 Gm.; 76 per cent polymorphonuclear leucocytes; 4 per cent large lymphocytes; 15 per cent small lymphocytes; 2 per cent monocytes; 1 per cent eosinophiles; 2 per cent basophiles. Red blood cells and platelets were normal. The blood was typed so that in case of excessive hemorrhage from the operation the patient could be given a blood transfusion during or immediately after the operation.

On Dec. 20, 1944, subtotal excision of the mandible was performed. Under pentothal sodium intravenous anesthesia with an intratracheal tube inserted to administer gas and oxygen and to prevent aspiration of fluid and blood from

the mouth, an incision was made along the gingival margin from the left lower second molar to the right lower first molar. The mucoperiosteum was detached from the bone, and, by splitting the flap in the medial line, it was possible to expose the entire outer surface of the mandible, after detaching the levator menti muscle, the depressor labii inferioris and depressor anguli oris muscles on both sides, and the buccinator muscle on the left. The surface of the



Fig. 408.—Photograph showing the expansion of the outer surface of the mandible.

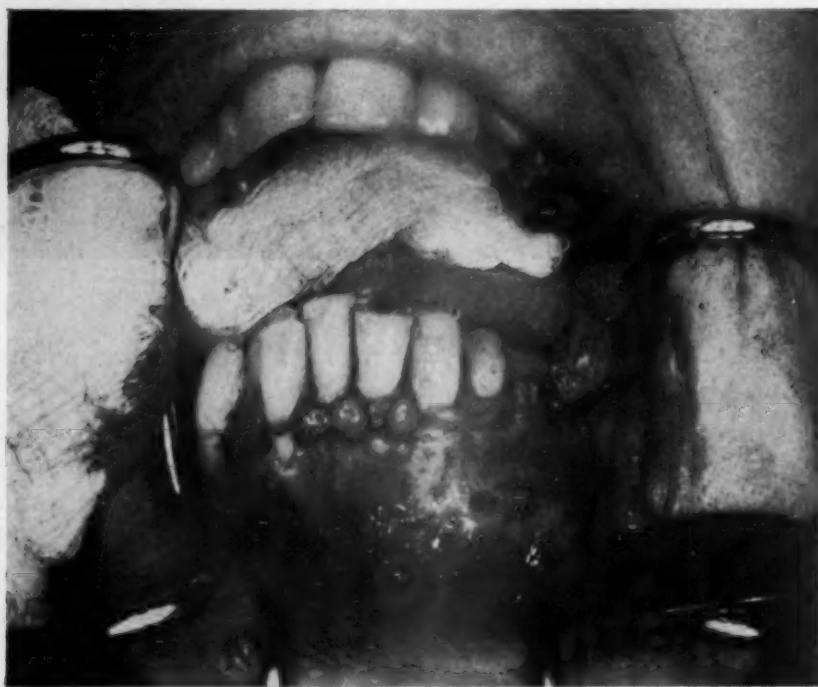


Fig. 409.—Photograph showing the exposure of bone after retracting the mucoperiosteum.



Fig. 410.—Photograph showing the mandible after extracting teeth and incising the bone.

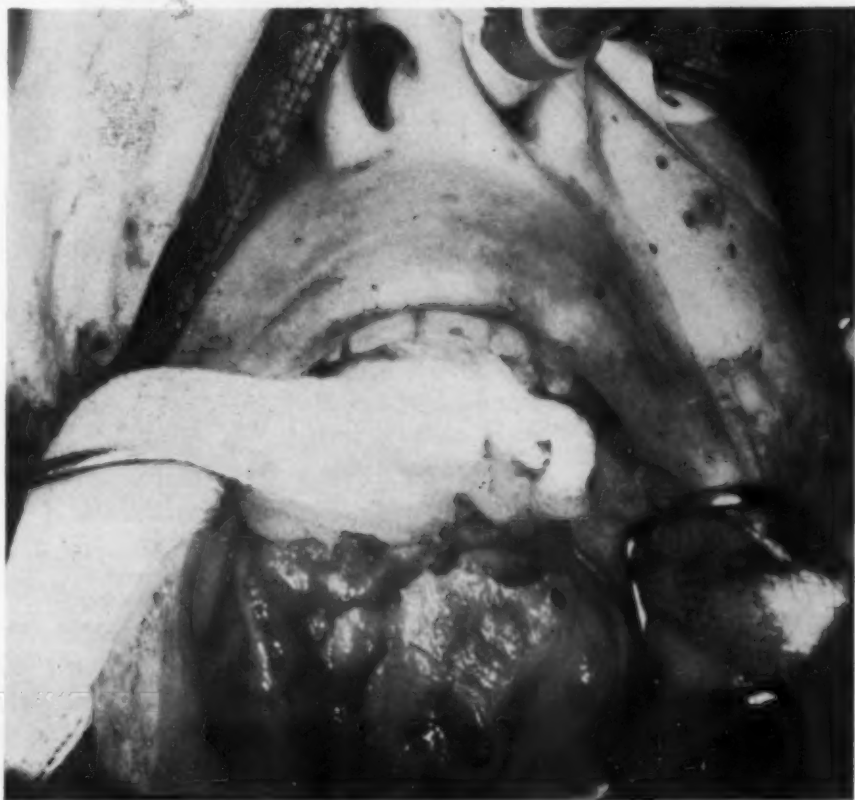


Fig. 411.—Photograph showing outer plate of mandible ready to be removed with tumor mass.

mandible presented evidence of localized expansion, especially in the region of the mental fossa and on the left, where the bone had a greenish appearance. There were no perforations (Fig. 409). All the involved teeth, namely, the left lower second molar, first premolar, canine, and four incisors, right canine, and first premolar, were extracted (Fig. 410). The interalveolar septa were cut with rongeurs, and by means of a dental drill, holes were made in the sound bone at the inferior border of the jaw to outline the region where the bone was to be osteotomized. On the left, similar holes were drilled in the sound bone posterior to the involved area, and on the right just anterior to the second premolar. By means of an osteotome, the drill holes were connected, and thus the entire outer surface of the jaw could be detached and removed with the tumor attached (Fig. 411). Active bleeding occurred in several places, particularly in the region of the mandibular canal on either side, and in the median line.



Fig. 412.—Pressure bandage to hold skin in contact with the bone, and prevent hematoma from forming.

These places were packed with bone wax temporarily. The tumor tissue attached to the bone had a pearly white appearance, and there were several large cysts. Next, the bone was carefully inspected for extension of the tumor into small accessory chambers. Three of these were found, and the tissue removed with a curette. Then the bone was carefully trimmed, both the alveolar bone and the inferior border of the jaw, and made smooth with a bone file. The bone wax was removed, after which the bleeding recurred; it was arrested by placing fibrin foam saturated with thrombin over each area. The mucoperiosteum was



replaced and sutured with tantalum 0.007 sutures. A pressure dressing was applied to the chin and held with a Barton bandage to obliterate space and bring the mucosa and subcutaneous tissue in contact with the bone (Fig. 412). The patient left the operating room in good condition. Because she had lost considerable blood during the operation, a transfusion of 500 c.c. whole blood was given.

On the first postoperative day, hydrogen peroxide mouthwashes, one-half strength, were used every two hours. The mouth was cleaned and the wound painted with methylene blue on each successive day. The patient made an uneventful recovery, and was discharged on Dec. 30, 1944, to be followed at the office.

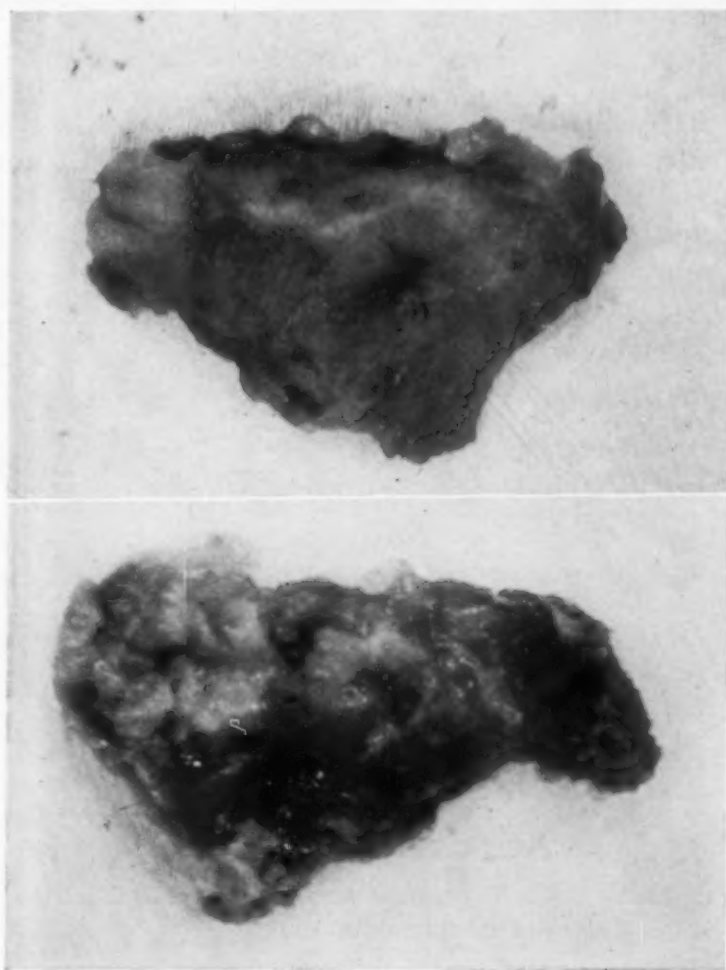


Fig. 413.—Excised specimen, outer and inner surfaces.

Pathologic examination: The specimen consisted of a shell of bone measuring 5 by 2.5 cm. (Fig. 413); it presented a mass of red gray, soft tissue on its concave aspect.

Microscopic examination showed in cross section a thin layer of bone with tumor tissue attached on the inner surface (Fig. 414). This tumor tissue con-

sisted of closely packed connective tissue cells and collagen fibers in which trabeculae of new bone and small cementicles had been deposited (Fig. 415).  
Diagnosis: Ossifying fibroma.

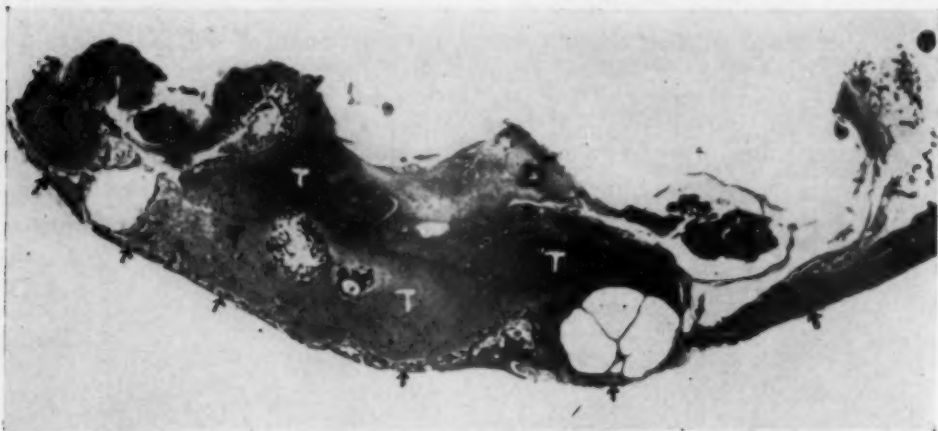


Fig. 414.—Low-power photomicrograph of a section through the ossifying fibroma. Arrows point to very thin outer cortex; T, tumor tissue.

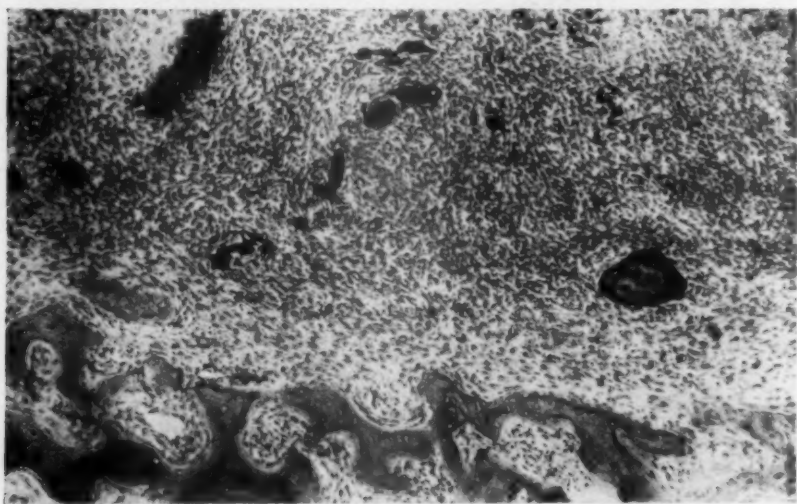


Fig. 415.—High-power photomicrograph of ossifying fibroma.

#### Case 88

##### Mixed Tumor of the Cheek

H. J. T. (292420), a 53-year-old man, was admitted to the hospital on Oct. 16, 1944, with a chief complaint of tumor of the cheek. This was first noticed one month previously when new dentures were fitted. The swelling had not bled and was not painful, but was tender to pressure. The patient had lost no weight.

Examination showed a tumor of slightly spongy appearance in the right cheek. It was ulcerated but not bleeding, firm, and slightly tender. It was about 2 inches long by 1 inch wide, and elevated about  $\frac{1}{2}$  inch (Fig. 416).

On Oct. 17, 1944, after the usual premedication and under endotracheal gas-oxygen-ether anesthesia, the oral pharynx was thoroughly packed and an

incision was made on the inner surface of the left cheek, extending from the corner of the lip to the ramus. The mucosa was dissected away from the tumor mass on both sides, and the mass grasped with a double hook and removed by dividing the surrounding structures by sharp and blunt dissection. The



Fig. 416.—Mixed tumor of buccal mucosa.

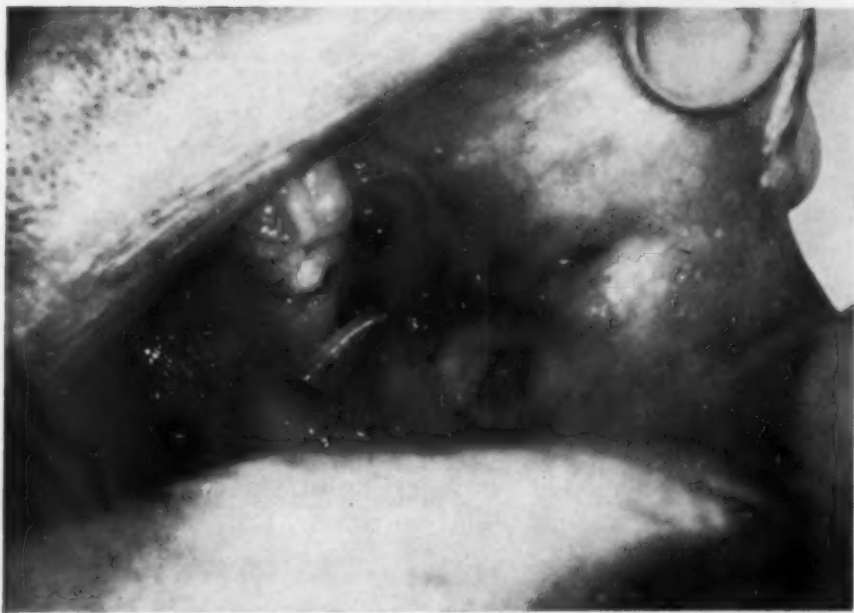


Fig. 417.—Incision closed and rubber-dam drain inserted after excision of tumor.

buccinator muscle was completely removed. The parotid duct was cut and the tissue removed as far back as the anterior border of the masseter muscle. After this the cavity was inspected and found to be free of all tumor tissue.

The incision was then closed with interrupted silk sutures, and a rubber dam drain was inserted in the posterior end of the incision, and attached to the mucosa with silk to establish a salivary fistula (Fig. 417).

Pathologic examination: The specimen consisted of a soft piece of red tissue with much fat and muscle attached, measuring 4 cm. in diameter. Diagnosis: Mixed tumor of the salivary gland type (Fig. 418).



Fig. 418.—Photomicrograph of section of mixed tumor.

The postoperative course was uneventful, and the patient was discharged from the hospital on Oct. 23, 1944, to be seen at the office for regular checkup examinations. On Nov. 20 a recurrence of the tumor was noted, extending under the zygomatic arch. He was again admitted for operation.

Under pentothal sodium intravenous anesthesia with an intratracheal tube inserted through the nose, the tumor tissue was removed by electrocautery. The cavity which extended under the zygomatic arch was packed with a boric strip.

The microscopic examination of the excised tissue again showed a mixed tumor of the salivary gland type.

The patient was discharged three days later, to be seen at the office. On December 4 the wound had completely healed, and on another checkup four months later there was no evidence of recurrence.

#### Case 89

##### Adenocarcinoma of the Palate

M. W. (468074), a 46-year-old woman, entered the hospital on Oct. 28, 1944, complaining of a tumor of the palate.

The patient had had the tumor for the past fifteen years. There had been no change in size, no pain, discharge, nor bleeding (Fig. 419). Two weeks



before, the tumor had been biopsied, and since then it had been "festering." There was no history of high blood pressure, cardiac or respiratory diseases. The biopsy material was diagnosed as mixed tumor of the palate (Fig. 420).

Examination showed a discrete mass about 2 inches in diameter and raised about  $\frac{1}{2}$  inch on the right side of the hard palate at the junction with the soft palate.



Fig. 419.—Mixed tumor of the palate.

X-ray examination showed a diffuse haziness of the left maxillary sinus. The remaining paranasal sinuses appeared to be normally aerated, but the ethmoids also showed a little thickening of the membrane on the left side. The nasal septum was devitalized on the right and showed a spur. The frontal sinuses were normal. There was evidence of infection of the left maxillary sinuses and probably the left ethmoids.

On Oct. 21, 1944, under pentothal sodium intravenous anesthesia, an intra-tracheal tube was inserted through the nose. The oral pharynx was packed with gauze. By means of an endothermy loop the uvula and right side of the soft palate were excised and the tumor tissue on the hard palate as well. The wound was carefully inspected for remains of tumor, and the bleeding stopped by coagulation. The patient left the operating room in good condition.

Pathologic examination: The specimen consisted of many soft, reddish-gray, irregular fragments of tissue measuring up to 1.5 cm. The edges were seared black; the surface was gray, granular, and the tissue meaty. The tumor, although slow growing and well differentiated, was definitely invasive (Fig. 421). Diagnosis: Adenocarcinoma.

The palate was painted with methylene blue for about a week. It was decided that because of the unexpected invasive character of this tumor and its malignancy, the patient should receive x-ray treatment. In preparation for this, the right maxillary second premolar and canine, and the second and third molars were extracted with local anesthesia, and, on another day, the right lower

premolars and molars. The patient had some soreness in the throat, but this was decreasing, and the tissue on the hard palate was beginning to granulate. The patient was discharged on Nov. 6, 1944, and on the same day x-ray treatments were started. She received 300 r. daily for eighteen days with the million volt

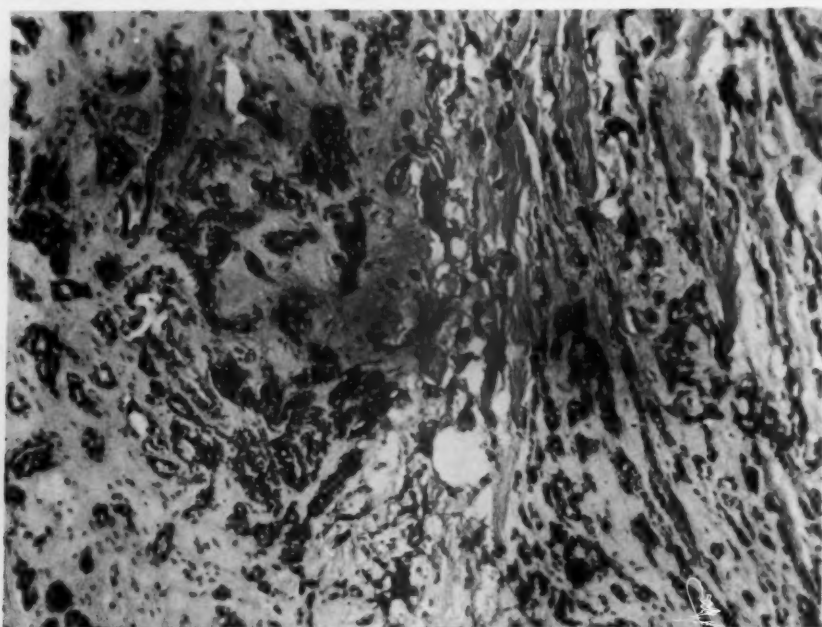


Fig. 420.—Photomicrograph of biopsy from hard palate, showing mixed tumor.

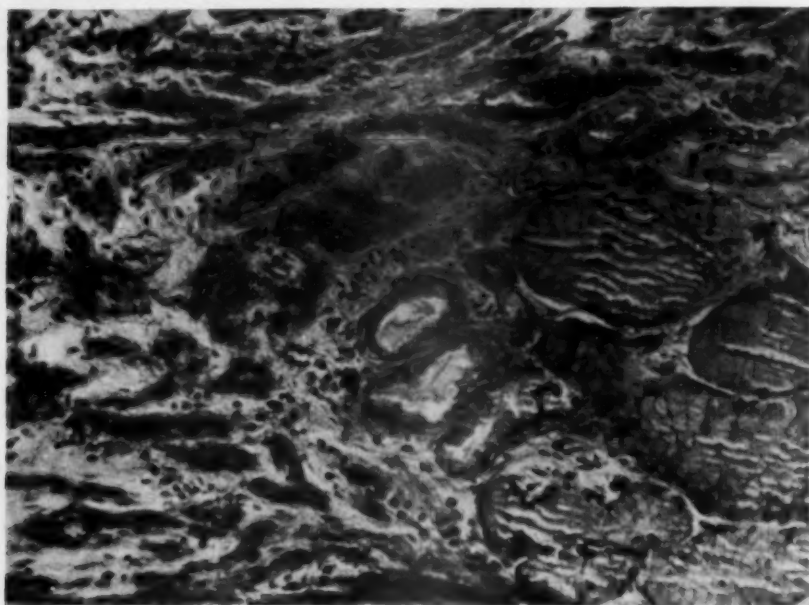


Fig. 421.—Photomicrograph of excised tumor of soft palate, which proved to be adenocarcinoma.

machine, a total of 5,400 r. She had a severe skin reaction and a large slough on the palate and pharynx, which gradually was thrown off. She is being examined at regular intervals for recurrence.

**Case 90****Carcinoma of the Mandible**

A. H. (473705), a 71-year-old man, entered the hospital on Dec. 13, 1944, for the excision of an ulcerative lesion in the lower jaw. The patient associated the origin of the trouble with irritation caused by a loose lower denture starting two or three years ago. At that time the area over the gingiva was sore, raw, and white in color. This was thought to be a canker sore. The patient had not worn the denture since last June when the three lower teeth were extracted. The lesion had not changed in appearance recently, but he had lost about 20 pounds during the last year. Last month a biopsy proved the lesion to be malignant; it was diagnosed as an early epidermoid carcinoma. He was advised to have it excised, but delayed the operation for personal reasons. The patient's mother had died of cancer of the liver.

Examination showed an ulcerative area on the central part of the lower gingiva just left of the midline. It had a whitish covering in the center surrounded by reddish, irregular margins (Fig. 422). There was no great elevation of the borders, no nodules could be felt, and there was no active bleeding. Lymph nodes were not palpable either in the sublingual or submaxillary region, or in the neck.

Blood examination showed red cell count 4,310,000; white cell count, 6,500; 60 per cent polymorphonuclear leucocytes; 12 per cent large lymphocytes; 17 per cent small lymphocytes; 5 per cent monocytes; 4 per cent eosinophiles; and 2 per cent basophiles. Red blood cells and platelets were normal.

X-ray examination showed a small erosion in the alveolar process left of the midline, which was thought to be caused by invasion of the cancer tissue (Fig. 423).

On Dec. 14, 1944, under intravenous pentothal sodium anesthesia, a subtotal excision of a section of the mandible was performed. After the usual preparation of the face and mouth, the area involved by the carcinoma and ulceration along the alveolar ridge was incised with the diathermy knife, about  $\frac{1}{2}$  cm. in good tissue, first on the labial side and then on the lingual side. Since the lesion was in close proximity to the sublingual gland on the left, it was dissected out, leaving it attached to the bone. With a circular saw a vertical cut was made on the left side beyond the area affected in the region of the second premolar. On the right, approximately in the region of the canine, a similar cut was made. The lip was divided, and, by means of a drill and osteotome, the mandible was osteotomized in a horizontal plane. In this manner a block of bone with the pathologic tissue attached could be removed. Several bleeding vessels were tied, and the mucosa drawn over part of the bony wound. The lip was closed with silk sutures on the oral side and interrupted dermalon sutures on the cutaneous side. A boric strip was inserted for drainage through an opening into the floor of the mouth where the sublingual gland had been removed. The specimen was examined, and it was found that nodules were attached to the lingual surface of the bony segment.

Postoperatively there was some submandibular swelling and considerable slough in the incisor region. Methylene blue and saline irrigations were applied. The patient was discharged on December 23, improved, to be re-examined at regular intervals.

Pathologic examination: The specimen consisted of a section of bone measuring 3 cm. by 1.5 cm. with tissue attached to each side. Transverse sec-



Fig. 422.—Ulcerative lesion of alveolar process and floor of mouth, which proved to be epidermoid carcinoma on biopsy examination.

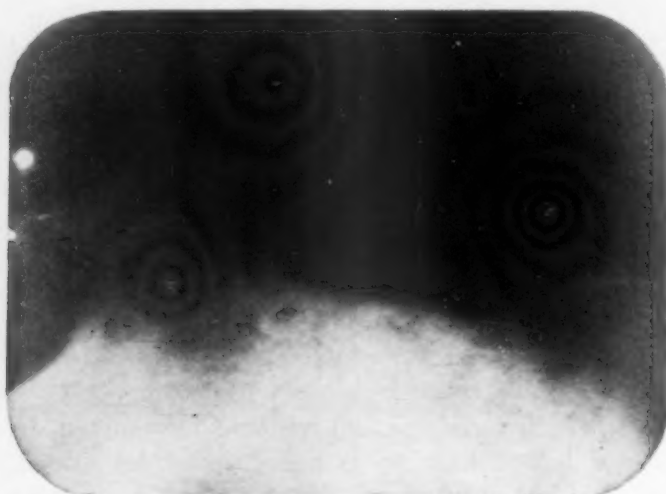


Fig. 423.—X-ray of alveolar ridge showing osteolytic area which was thought to be due to destruction by carcinoma, but which proved to be the unhealed extraction wound.



tions were cut in three places. In one section there was no carcinoma present; in the other two there were areas where the epithelium was absent, and areas where there was a malignant downgrowth from the rete pegs (Fig. 424). The



Fig. 424.—Photomicrograph of biopsy from ulceration showing early epidermoid carcinoma.



Fig. 425.—Section through excised part of mandible showing fibrous tissue in extraction wound, *E*.

bone itself was not involved. The osteolytic area seen in the x-ray was caused apparently by extraction of the teeth, and was filled with connective tissue (Fig. 425). Diagnosis: epidermoid carcinoma of the alveolar mucosa.

**Case 91****Large Carcinoma of Cheek**

IRA T. NATHANSON, M.D.

H. B. (462464), a 75-year-old man, was admitted to the hospital on Sept. 5, 1944, with a tumor of the neck.

The patient had a tumor in the right neck region under the ear. It was first noted about one year ago. Since that time there had been a steady increase in size. About three weeks ago the patient became unsteady on his feet and fell several times. Nine days ago he fell and struck the mass, causing it to bleed profusely. Since then there has been an ulcerated area on the mass.

Examination showed the patient to be undernourished and almost emaciated in appearance, with a sallow complexion. On the right side of the neck was a mass 15 by 10 cm., purplish red in color, extending from below the ear to the base of the neck overlying the sternocleidomastoid muscle, and extending anteriorly over the ramus of the jaw (Fig. 426). The mass seemed fairly movable; there were no lymph nodes or other masses felt. Because of the tumor the patient had some limitation of motion and difficulty in swallowing.

The white blood count was 11,800; hemoglobin, 10.1 Gm. In preparation for the operation the patient was given a high caloric, high vitamin diet. On September 9 he was given an intravenous infusion of 1,500 c.c. of 5 per cent dextrose with 2 ampoules betalin and 1 Gm. cevalin, and a transfusion of 500 c.c. whole blood. He also received 1 c.c. liver concentrate intramuscularly, 4 grains ferrie sulfate, and 4 mg. hypinone, daily. On the next two days he received 1,500 c.c. of 5 per cent dextrose in water, plus vitamins, and 1,000 c.c. of 5 per cent amigen in dextrose, intravenously. On September 11, in addition to the dextrose infusion he received 500 c.c. whole blood. On September 12, the day of the operation, the nonprotein nitrogen was 64 mg. per liter.

Excision of the tumor was performed under novocain anesthesia. After the usual preparation of the skin with ether, alcohol, and iodine, an incision was made in the skin at the anterior border of the lesion about 2 cm. away. By sharp dissection it was found that a line of cleavage could be obtained, and, therefore, the incision was carried around the entire tumor mass, including the lower half of the lobe of the ear. Many vessels were encountered in the course of the dissection, all of which were divided and individually ligated whenever possible. Considering the vascularity of the tumor and its large blood supply, there was a relatively small amount of gross bleeding because of the isolation of the vessels. The cut edges of the lobe of the right ear were approximated with interrupted silk and the skin edges were sutured to the raw space to obliterate as much as possible of the defect. The final defect was 10 by 12 cm. This patient was an extremely poor risk, but he withstood the procedure surprisingly well. Boric strips were applied to the defect, and a pressure dressing. The patient left the operating room in fair condition. He was given a transfusion of 500 c.c. whole blood, followed by 1,000 c.c. dextrose in saline.

The next day the patient seemed to be holding his own. He was given an intravenous infusion of 1,500 c.c. dextrose with 1 Gm. cevalin and 2 ampoules



Fig. 426.—Large epidermoid carcinoma at angle of jaw.

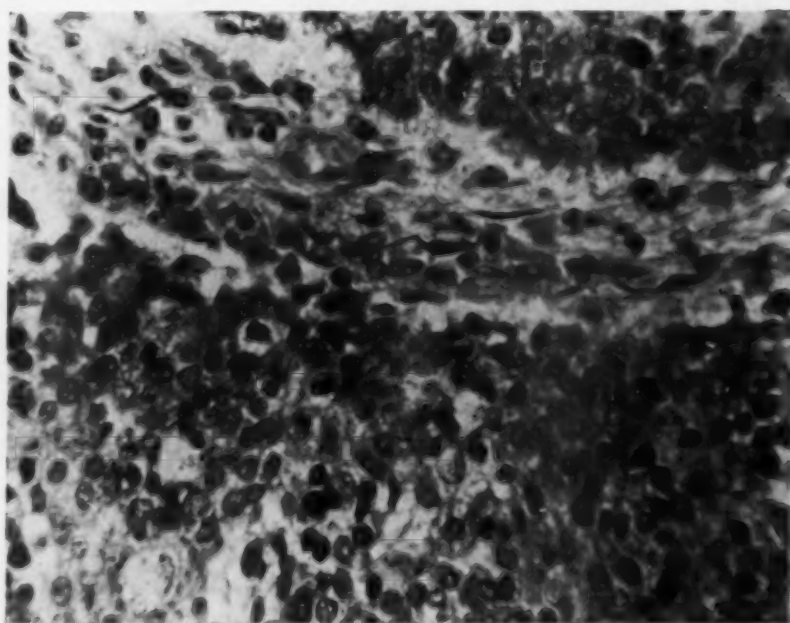


Fig. 427.—Photomicrograph of carcinoma, undetermined type.

betalin, and 500 c.c. amigen, 1 c.c. liver concentrate intramuscularly, and 4 grains iron; this was repeated on September 14. On September 15 the patient's condition changed for the worse; he had Cheyne-Stokes respiration; the temperature, pulse rate, and respirations were all elevated. The nonprotein nitrogen was 52 mg. per liter. He was given 2 c.c. cedilanid intramuscularly. In spite of all efforts, however, the patient died on September 16.

Pathologic report, gross: a hemispherical mass of friable, moderately soft, gray-brown meaty tissue, measuring 12 cm. in diameter and 7 cm. high, was covered on the round surface with skin that was partially covered with hair, and in some areas was thin and irregular with many sessile and pedunculated nodules measuring up to 2 cm. in diameter. The flat amputation surface was red and covered with a thin layer of fascia. The tumor tissue extended everywhere to the amputation surface. On section the mass had a 2 cm. broad capsule of light gray meaty tissue within which was a yellow-pink mass of soft necrotic tumor tissue. Diagnosis: Carcinoma, type undetermined (Fig. 427).

### Case 92

#### Unilateral Parotid Swelling

DAVID WEISBERGER, D.M.D., M.D.

H. S. (474528), a 21-year-old woman, entered the hospital on Jan. 9, 1945, with a swelling on the left side of the face.

About two and one-half months ago the patient was delivered of her second child without complications. Five days later a swelling in the left preauricular area was noticed; this gradually enlarged and became tender. The whole side of the face was involved and the eye almost closed. The swollen area became painful, and the skin red and shiny. The face was numb and the patient could not move the left side; she had difficulty in opening the mouth to eat. Originally the pain had been limited to mealtime, and was present under the left mandible. About one and one-half months ago the swelling reached its maximum. During this time the patient had a fever for about ten days. She had been in another hospital for two weeks where sulfonamides and penicillin were given, and incision and drainage were performed twice at about a week's interval. About one month after the swelling on the left appeared, the right side started to swell. There was a small mass in the preauricular area, but this never enlarged over a few centimeters and was not painful and not tender. It disappeared while the patient was in the hospital. Shortly after the last incision and drainage, a small, red swelling appeared on the left side below the site of the drainage, and has persisted to the present time, although it has gradually decreased in size.

The patient's general health has been good, although she had headaches, dizzy spells, and tinnitus. Last summer she had a watery discharge from the nose when she bent over.

Examination showed a small, 2 by 3 cm., oval, fluctuant swelling in the left preauricular area. The edges overlying the skin were inflamed, and the central area was yellow in the most fluctuant part. There was only slight tenderness. The parotid tissue seemed to extend several centimeters beyond the edges of the raised mass, and felt indurated. There was a scar from the previous incisions. The ducts in the mouth were normal; no pus could be expressed.



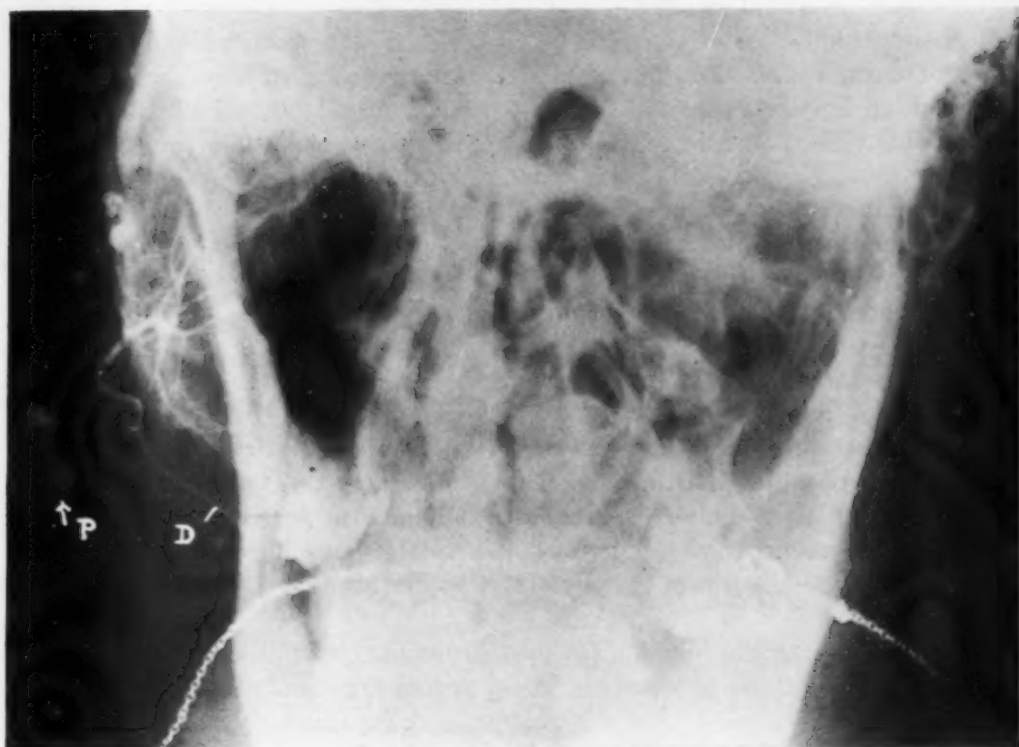


Fig. 428.—Sialogram of parotid gland showing several dilated pockets filled with lipiodol. *D*, Parotid duct; *P*, pocket filled with lipiodol.



Fig. 429.—Exaggerated Water's position. Sialogram showing dilated pockets filled with lipiodol. *D*, Parotid duct; *P*, pocket filled with lipiodol.

Impression: Bilateral parotid tumor. Sialograms were advised.

A sialogram taken on January 10 showed the left parotid gland and duct to be of normal size. The lateral and anterior portion of the gland showed several dilated pockets consistent with sialectasis (Figs. 428 and 429).

At this time the white cell count was 8,850; hemoglobin, 96 per cent; prothrombin time, 17 seconds, normal 18 to 20 seconds; serum amylase, normal.

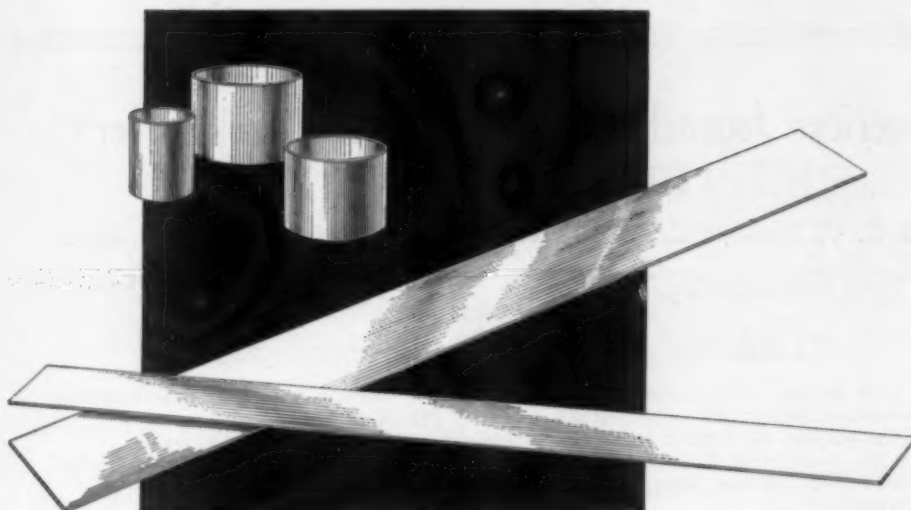
On January 16, 4 c.c. of grayish-yellow pus was aspirated from the abscess cavity through normal tissue. The culture showed *Staphylococcus albus*. On the next day 4 c.c. of grayish fluid was again aspirated, and the cavity collapsed. It was irrigated with 4 c.c. penicillin. The patient tasted a "salty" fluid in the mouth, and the fluid was reaspirated and a pressure dressing applied. X-ray examination showed that the lipiodol previously injected into the left parotid gland had entirely disappeared.

On January 18 the patient was shown at grand rounds, and it was decided that a regime of multiple aspirations with penicillin therapy should be followed. Two days later after dinner the patient complained of severe headache on the right side and vomited once or twice. She had blurring of vision. She stated she had had similar attacks previously. Codeine brought relief. Impression: Migraine.

On January 25 the patient was seen in dental consultation. Examination of the secretion of the left parotid gland revealed a scanty and viscid saliva which could be seen only after stimulation upon chewing a lemon. The duct was probed with various-sized lacrimal-duct probes, and it was found to be very narrow and constricted. An attempt was made to dilate the duct. Following this, stimulation with lemon juice produced a moderate increase in thin saliva. It was believed that the gland would never function normally.

The patient was discharged on January 25 to be followed in the Outpatient Department for dilation of Stensen's duct.

On February 1 the patient said the left side of the face was better. The duct was probed, but very little saliva was present.



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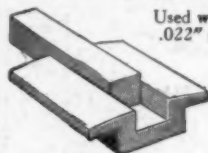
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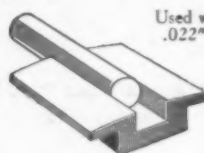
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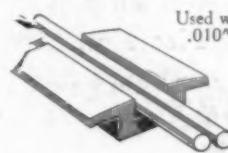
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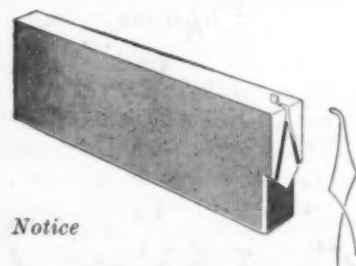
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# Northwestern University

## OSTEOGENIC SARCOMA OF THE MANDIBLE

### A CASE REPORT

FREDERICK W. MERRIFIELD, D.D.S., M.D.

THE bone structure of the body is affected by injury, infection, nutrition, and new growths.

The bones of the jaws are no exception, but they show a tendency to resist disease better than other bones. Osteomyelitis of the mandible is rarely the intractable type of disease that it may be elsewhere. Osteogenic sarcoma of the mandible, the subject of this report, is a rare disease compared to its incidence in other bones, notably the tibia, femur, and pelvis.

Ewing, Codman, Geschickter and Copeland, Waldron, and others have devoted themselves to the problem of the classification of bone tumors and have made great progress in this regard. The difficulties were well expressed by Homan. "Since bone, which is mesoblastic in origin, is perhaps the most highly finished product of the primitive fibroblast, it is not surprising that the tissue of its tumors would vary from the most primitive to the most differentiated, and that, according to the general rule, new growths of bone should be, on the one hand, remarkably malignant and on the other, almost completely benign. There is, moreover, a most confusing variety of appearances in any one tumor. Thus a classification of these tumors is difficult." All authorities are in agreement that true tumors of jawbones, per se, are rare. Padgett emphasizes the relationship between normal bone development and tumor formation and gives reasons based upon this observation for the scarcity of true bone tumors in the bones of the jaw. All bone components are derived from preformed connective tissue. In nonmembranous bones, fetal cartilage cells give rise to adult cartilage cells, and this adult cartilage is ossified by a gradual process and true bone is formed. In the early stages of either variety of bone formation, new growth is unlikely to occur. It is in bone formed from cartilage, in which the transition steps are delayed over a longer period of development, that tumor formation in bone is likely to occur.

Specifically, the reasons for the variety of tumors of the jawbones proper, based upon the foregoing observations, are that the bones of the jaws are predominately membranous. The areas of cartilagenous origin in the mandible, with one exception, pass through the cellular transition completely and are calcified by the one of the first year. The exception is the epiphysis of the condyle which becomes calcified at about the fifteenth year.

Osteogenic sarcoma is most likely to appear in the second decade of life, in otherwise strong and healthy young persons when intensive development of bone occurs.

Trauma, as an exciting factor in the disease, has been the observation of most authors. Kolodny believes that 70 per cent of patients over 10 years of age have such a history. Ewing, however, analyzes the etiological factor of trauma and draws particular attention to the proliferative activity of fibroblasts, osteoblasts, and endothelium which can be separated from sarcoma only with difficulty, noted in the callus of healing bone injuries. In this connection, we have knowledge of tissue from a subperiosteal swelling of the mandible, following extraction of an infected tooth, reported on frozen section as osteogenic sarcoma. The jaw was resected, and a later report on the tissue removed at operation was: "Inflammatory tissue." Ewing is of the opinion that while all varieties of sarcoma have been attributed to trauma, injury is only one of the essential factors. He believes that "many or most cases" fail to give a history of trauma, that many tumors arise in bones protected from injury, and that occasionally bone sarcomas are multiple.

The mandible is probably the most frequently injured of the facial bones, but osteogenic sarcoma occurs less frequently here than in the maxilla by the proportion of one to two, and Christensen's classification of one thousand bone tumors includes eleven osteogenic tumors in the maxilla and only seven in the mandible.

The clinical diagnosis of osteogenic sarcoma may be difficult. Persistent, increasing, and unexplained pain is the earliest symptom and one which may be overlooked. The aching, boring character of the pain, which is worse at night, is similar to the onset of osteomyelitis of the jaw. In osteomyelitis, however, there is associated dental disease, which is usually absent in sarcoma. The symptoms may be present some time before relief is sought. Mr. R. E., the subject of this report, had teeth extracted for relief of "toothache." The clinical findings vary greatly with the particular type of tumor. In this case, the facial contour was changed without skin invasion, but the growth was rather slow; the general condition of the patient was excellent and there was no evidence of lung invasion. There was submaxillary lymph node enlargement, but of inflammatory type. X-ray examination, as is usual, was somewhat confusing and films of diagnostic value were obtained only after repeated effort. The report on the first film was "osteomyelitis of mandible."

#### CASE REPORT

*History.*—Mr. R. E. Until two years ago, the patient was well and healthy, at which time he noticed a pea-sized swelling on the gum of the lingual surface of the lower left cuspid. Prior to this (three to four months), the patient's pipe (in his mouth) was hit by a log chain from a winch on a truck. The pipe stem was driven against the labial surfaces of the incisors and cuspid on the left side, driving them loose. These teeth straightened out spontaneously and apparently returned to normal. No other exciting factors were recalled.

One month after the lesion was first noticed, it ruptured spontaneously, releasing a yellowish thin fluid. At this time, there was no increase in size. This ulceration healed quickly. From this time until April, 1943, the lesion gradually increased in size. In April, the patient had all of his lower teeth removed (against the advice of his dentist) because he said that they were "badly decayed and pieces chipped off easily." There were apparently no complications immediately following the extractions.

Three to four weeks after the extraction, the swelling began to increase, becoming visible on the exterior of the left cheek. The lesion progressed slowly in size until the patient went to a dental clinic. At the clinic, the patient states that a biopsy was taken and he was informed the diagnosis was "sarcomic tumor." The patient continued on at the clinic

for two months, refusing operation, which was advised. The jaw did not improve and sequestra of "bonylike" substance up to about 1 cm. in size began to be expelled from the lesion. It was uncomfortable, but not painful, at the time these pieces were extruded. The patient has removed several pieces himself with tweezers. A moderate amount of bleeding was the only consequence of each removal. The only discomfort or pain reported was a feeling occasionally as though "a pin were sticking him." This pain "is not severe, may occur at any time, and almost any place in the region of the jaw on the left." On Jan. 31, 1944, the patient was referred to the tumor clinic of Northwestern University Medical School.

*Examination.*—A well-developed, well-nourished white man, aged 38 years, not acutely ill. The patient was found to be in fairly good general health, with nothing of any significance except in the mouth. Blood and urine examinations were normal. Kahn and Wassermann reactions were negative.



Fig. 1.

Examination of the mouth revealed a diffuse, hard and slightly tender mass involving the left side of the mandible, from the cuspid to the second molar areas. The gums showed marked hypertrophy and the continuity of the mucosa was broken in several places by crater-like ulcers with gritty bases and containing gritty material. The upper teeth were carious and covered with tartar on the left side. The lower teeth had been removed. The left side of the face was moderately enlarged, but the skin was unaffected. In the neck, the left upper anterior cervical nodes were palpable, rather soft, and freely movable. Smaller nodes were palpable on the right side.

Report obtained on the original biopsy was verified. The diagnosis of "osteogenic sarcoma" was made.

*X-Ray Examination.*—Jan. 24, 1944. "The films of the left jaw region show a great amount of soft tissue swelling which was observed clinically. In addition, there is an enlargement of the body of the mandible on the left. The radiating new bone formation extends superiorly into the soft tissues of the alveolar surface giving this lesion a malignant aspect and an invasive character. New bone formation suggests osteogenic sarcoma." (Fig. 1.)

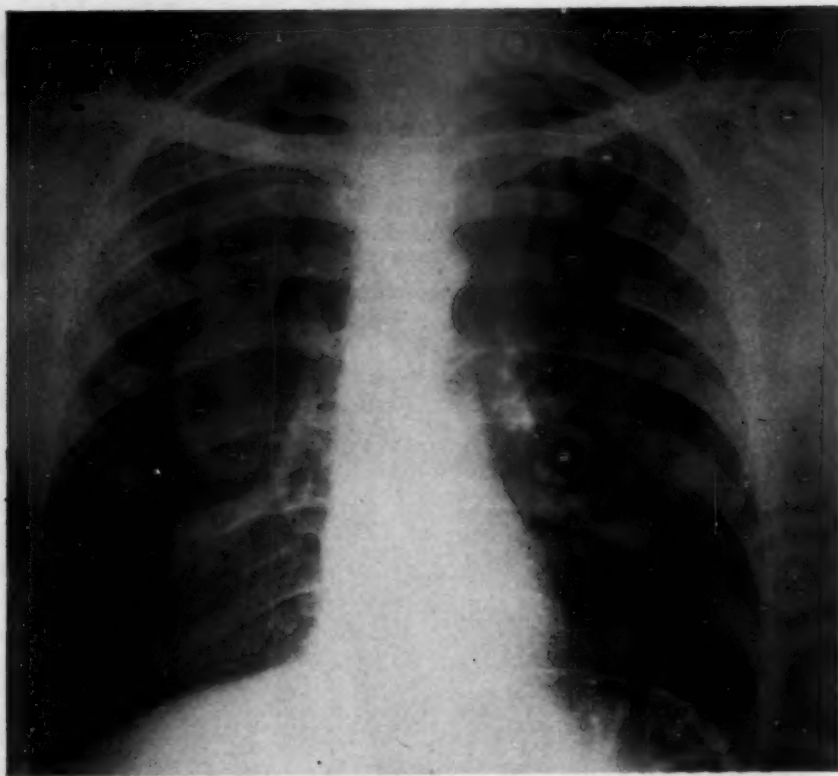


Fig. 2.



Fig. 3.



"Impression: Malignant bone tumor, body of left mandible" (A. F. Galloway).

Jan. 24, 1944. "Chest films showed a healthy chest with no evidence of metastasis to bone or soft tissue structures" (Fig. 2) (A. F. Galloway).

Jan. 31, 1944. "Re-examination of the jaw shows the same findings more clearly than were observed on the previous film study. The opinion remains the same" (A. F. Galloway).

Impression: Favors osteomyelitis rather than malignancy (Dr. "C").

Impression: Not convinced films indicate malignancy (Dr. "B").

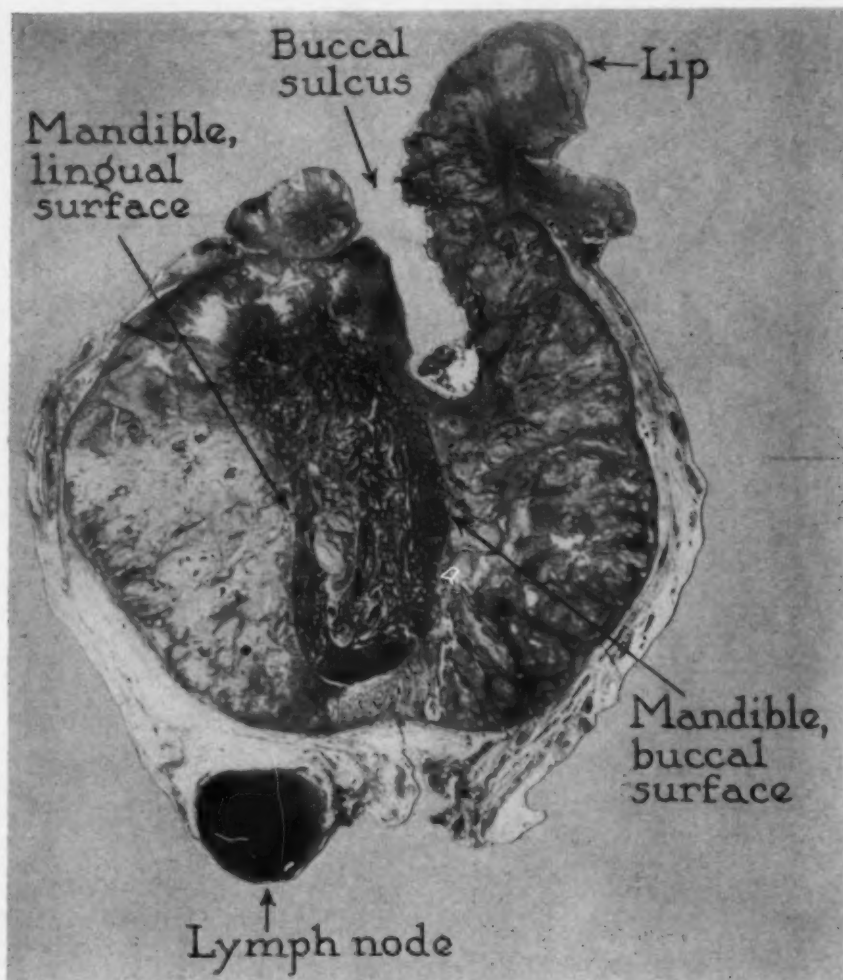


Fig. 4.

*Operative Note.*—The patient was operated upon on Feb. 17, 1944, under avertin anesthesia. The left side of the face was exposed. A midline incision was made in the inferior labium, carried to the inferior border of the mandible and then posteriorly to the angle of the jaw. A flap was made by sharp dissection and turned back. The jaw was cut through at the midline and also through the ramus above the angle, with a Gigli saw. The mass was freed from the floor of the mouth and removed. There was no undue hemorrhage. The facial artery and vein were the only vessels ligated except for the usual small bleeders. The intra-oral mucous membrane was approximated where possible and the skin incision was closed with black silk. A Penrose drain was inserted and a moderate pressure bandage applied.

*Pathologic Report.*—Gross Description: "The gross specimen (Fig. 3) consists of a portion of the mandible, 9 cm. in length, together with surrounding tissues. The mucosal surface is edentulous and has a nodular irregular appearance. Near the posterior end of the



Fig. 5. (Magnification  $\times 50$ .)

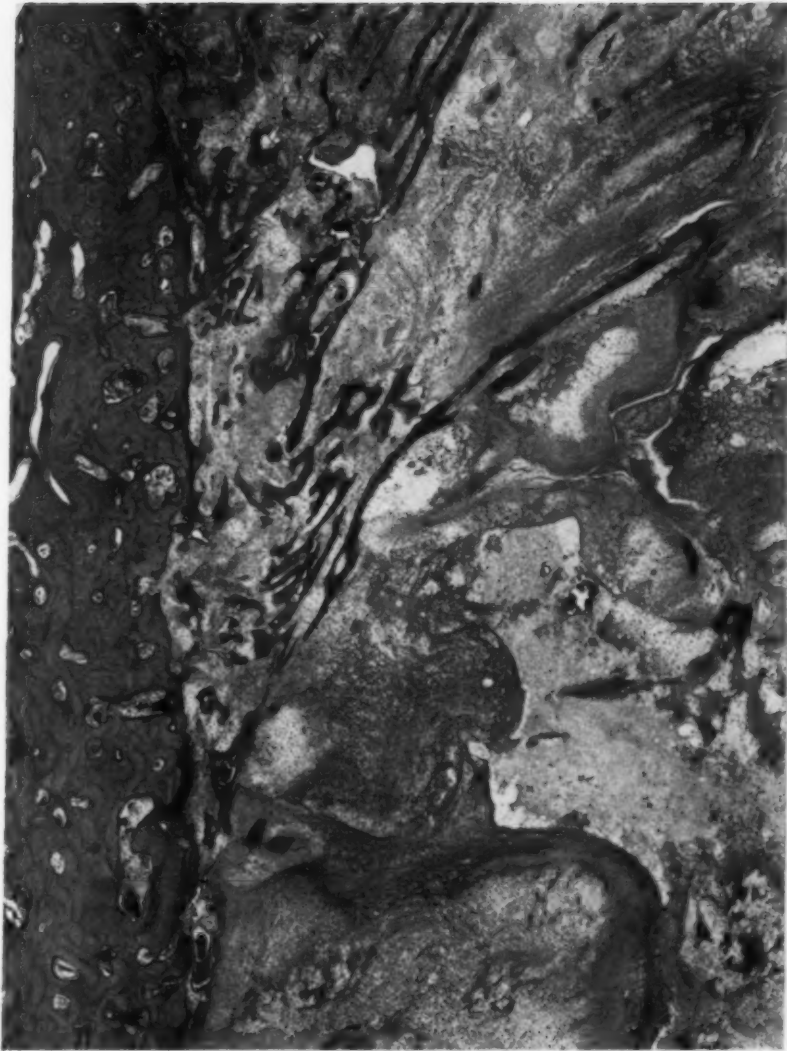


Fig. 6. (Magnification  $\times 150$ .)

bone, there is a nodular round swelling  $4\frac{1}{2}$  by 4 cm. in diameter. On section through this area, the appearance is that of a firm fibrous tumor having a radial structure arising in the periosteum. The bone itself does not appear to be greatly affected.

Microscopic: "Three blocks, six sections. The first block (Fig. 4) is partially covered with the uncornified stratified squamous epithelium of the mouth, showing marked elongation of the rete pegs. Lymphocytes and plasma cells are numerous in the subcutaneous tissue. The deeper portions are composed of tumor tissue, consisting of osteoid tissue containing spicules of newly formed bone (Fig. 6). Occasional masses of cartilage are present, some of which show myxomatous change. The basal portion is covered with granulation tissue and purulent exudate.

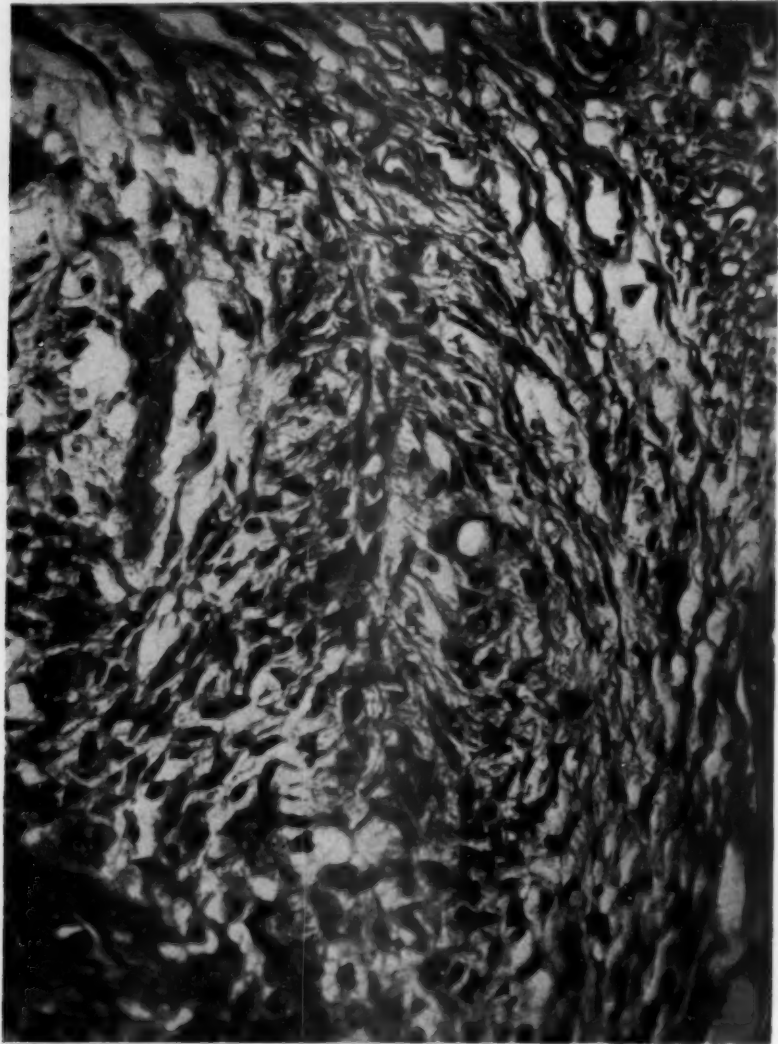


Fig. 7. (Magnification  $\times 400$ .)

"The second block (Fig. 5) is also composed of osteoid tissue and myxomatous cartilage. Occasional areas of calcification are present in the osteoid tissue. Some voluntary muscle and fibrous tissue are present on the outer surface. The tumor appears to be well encapsulated at this point and does not invade beyond the capsule.

"The third block (Fig. 7) consists of a small lymph node containing numerous small primary follicles. The sinusoids are choked with small lymphocytes and plasma cells. There is no evidence of metastasis in this section."



Diagnosis: "Osteogenic sarcoma of the left mandible; chronic lymphadenitis in regional lymph node."

Comment: "There is no evidence of metastasis" (D. O. Manshardt).

*Postoperative Course.*—There was an elevation of temperature for six days to a maximum of 102.4° F. on the first postoperative day, and becoming normal on the seventh day. Some of the skin sutures and the rubber drain were removed on the fifth day. The remaining sutures were removed on the seventh and eleventh days, respectively. There was drainage from the external wound for about three weeks, and small bone splinters were extruded. Complete healing then took place.



Fig. 8.

The patient reported for observation in January, 1945. The mucosa of the mouth had healed perfectly. The external wound was healed, but with a rather irregular scar, resulting from the prolonged drainage. Strangely enough, there was no deviation of the jaw to the operated side. This is unusual in a resected, edentulous jaw (see Fig. 8).

Function was good and the patient had no complaint relative to his ability to eat, and he was back at his usual work.

There was at this time no evidence of recurrence or of metastasis.

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## PRINCIPLES CONCERNING THE EXTRACTION OF TEETH

WARREN R. SCHRAM, D.D.S., M.S.D.

**I**F THERE is justification for the presentation, in this discussion, of material which is common knowledge, it lies in the fact that it is the fundamental principles put into practice in operative technique which produce favorable results.

The instrumentation used to induce local anesthesia is well standardized. Aside from the technique employed, the quality of the anesthesia is dependent upon the solution which is used.

We believe that the alkaline solution, which we have used at Northwestern Dental School over a period of many years, has certain advantages over one which is acid.

Freeman<sup>1</sup> states, "In an alkaline solution the dissociation of the procaine base is more rapid, and consequently the base is liberated and is free to act on the nerve tissue more promptly with a much more rapid induction time. This has been measured and recorded in the goldfish experiment, and has been repeatedly demonstrated in the clinic, with almost immediate anesthesia following injection.

"The use of an alkaline solution reduces the local tissue irritation very definitely, since the tissues are not called upon to neutralize the solution which is already approximately the same pH as the blood. The alkalinity of the solution probably also favors quicker recovery of any operative trauma, since the tissues have not been deprived of the normal alkalinity in neutralizing an anesthetic.

"There appears to be definitely less general systemic disturbance in the use of alkaline solutions, possibly because there is less shock in introducing a solution which conforms to the pH of the tissues. The marked decrease which we have noted in fainting and other systemic manifestations since we have used alkaline solutions may be due in part to the fact that we have learned to inject the solution more slowly, but we are also convinced that the alkaline solutions have contributed to this desirable freedom from general reactions.

"We should also mention the principal disadvantage of the alkaline solution, the fact that it is relatively unstable and must be freshly prepared to be most effective.

"We believe a freshly prepared solution is still the best available local anesthetic, although the manufacturers have made distinct improvements in their products in recent years.

"The aseptic precautions in the use of a local anesthetic should be very carefully guarded, and every operator should check the steps in preparation from time to time to see that no imperfection has been allowed to enter into his routine. The manufacturer may have delivered his product with every

<sup>1</sup>The Department of Oral Surgery, Northwestern University Dental School, Chicago, Ill.

aseptic precaution, but a careless moment in chairside preparation will undo all the scientific safeguards which have been employed."

With a well-worked-out plan of preparation, an alkaline procaine solution can be made fresh for each series of injections with little loss of time. This may be done by dissolving procaine hydrochloride tablets in a 0.1 per cent solution of sodium carbonate. Such a solution may be easily prepared by adding  $\frac{1}{4}$  Gm. of the anhydrous carbonate to 250 c.c. of distilled water. This stock solution is stable and may be boiled occasionally to insure sterility. The operator is well repaid for each refinement he is able to introduce into his technique and into the materials which he employs to induce local anesthesia. Profound anesthesia is one of the most important aids to good surgery.

All sound surgery is based upon tissue handling calculated to favor repair. Satisfactory repair following tooth removal requires that uneventful healing take place and that a tissue contour remain which is favorable to prosthetic restoration. Such repair is most likely if the operator's technique is directed by a sound knowledge of the behavior of bone and soft tissue as they react to mechanical and bacterial injury.

Mangos<sup>2</sup> states, "It would seem inadvisable to interfere with the alveolar bone in any way except to round off and smooth the sharp prominences. By so doing, as much ridge height and breadth as possible would be conserved, which factors contribute greatly to the stability of a denture.

"These principles, of course, would not apply in the case of an alveolectomy where it was necessary to remove excess bone for cosmetic purposes or for satisfactory denture adjustment."

The contour which remains after final healing has taken place can be controlled, to a large extent, by bone handling at the time of operation. From clinical experience it is known that, if sharp projections of bone are left in the area following tooth extraction, final repair is delayed. Histological studies show that these irregular prominences are slowly reduced by osteoclastic action. In this connection, however, it should be borne in mind that as this process goes on there is a filling in of depressed areas in the bone. Therefore, it may be desirable in selected cases to leave such prominent areas in order to encourage the building of a more satisfactory ridge when healing has taken place.<sup>3, 4</sup>

Boyd<sup>5</sup> says, "Owing to the readiness with which bone formation and bone absorption can occur, a *remodelling of bone* is continually taking place. Bone tissue is very sensitive to demands made upon it and responds readily to these demands. It is hardly too much to say that every change in the function of a bone is followed by definite changes in its internal architecture. As the result of a fracture the lines of stress in a bone may change as if it was one of the most plastic of structures."

If one were to select a patient in perfect health whose teeth and their supporting bone were free from pathology, inject an anesthetic solution which neither irritated the tissue injected nor slowed its circulation, and, finally, remove the tooth aseptically with no soft tissue or bone injury but simply by dividing the periodontal fibers, it is probable that rapid and certain healing would follow. The sphincterlike action of the fibers of the marginal gingivae would reduce the size of the orifice of the socket and favor the formation of a



firm satisfactory blood clot. Within a few days this clot would be organized into connective tissue to form a basis for bone repair, and its exposed surface would be covered by the proliferation of epithelium from the marginal gingivae.

Unfortunately, a tooth is usually removed because it or its supporting structure is infected. The solution injected for local anesthesia does slow the circulation at a critical time, particularly if its epinephrine content is high, and is not wholly without the potentiality of retarding repair within the field of operation. Thus, healing of a tooth socket takes place in tissues injured by infection and by surgical trauma, and perhaps also by the anesthetic solution.

In the consideration of the healing of extraction wounds the operator is concerned with the repair of soft tissue and bone. The reparative properties of the soft tissue of the mouth under normal conditions are great, and this tissue in itself offers little or no problem in healing. Bone, a derivative of connective tissue by adaptation, exhibits the same properties of repair as does soft tissue but to a markedly less degree. Because of its high content of lime salts, and because its nutrient vessels are confined in unyielding spaces and its blood supply is therefore precarious, it is more ponderous in its reaction to infection and in repair. In addition to this, portions of bone which are killed by mechanical or bacterial injury present the problem of foreign bodies in the wound.

A thoughtful consideration of the causes of faulty healing following tooth removal suggests that interference with the blood supply to the part is of great importance. One of the first rules of good oral surgery, then, is to leave sound bone well covered by soft tissue or blood clot. It should be borne in mind with the extraction of each tooth that the bone must be protected. Careful soft tissue handling will do this directly by providing a covering for the bone, and indirectly by helping to maintain a blood supply. A well-formed blood clot in a healing tooth socket is all-important since it performs the two functions of laying down a matrix for tissue growth and bone repair, and of protecting painful nerve endings during the process. The maintenance of a blood clot in the socket and its conversion into ossifiable connective tissue is brought about by the migration of blood vessels mainly from the bony wall of the socket. Therefore, it is evident that injury to the bone by burning, through the improper use of the surgical burr and the crushing force of instrumentation, makes the retention of the blood clot less likely. Alteration in blood supply in that critical period immediately following the removal of the tooth can be minimized by the slow injection of a local anesthetic solution with a low epinephrine content. A solution of 1:50,000 is recommended. Such a solution has an additional advantage in that its systemic effects are less.

In the operative procedure, if there is one thing which tends to minimize trauma, protect blood supply to the part, and to make success certain, it is to plan in detail, in advance, each step to be taken. The radiograph, the operator's knowledge of anatomy, and clinical examination of the part, all assist him to visualize structures with which he is dealing. In the clinical examination the character of the overlying soft tissue and bone, the presence of abrasion or carious destruction of the crowns of the teeth, and the degree of firmness of their attachment may be noted. The radiograph will show the structure and position of the roots, the character of the bone surrounding the



roots, and the presence of structures to be avoided, such as the maxillary sinus, the mandibular canal, the mental foramen, and the roots of adjoining teeth.

If it is possible to remove a tooth or root with forceps without undue injury to soft tissue or bone, that method is likely to be the least traumatic and consequently the one to be preferred. In certain instances it is advantageous to separate multirrooted teeth and remove each root separately by the use of forceps or suitable elevators. In cases of hypercementosed roots it is advisable to remove impacting bone by the use of the bibevel drill. This instrument may also be used to gain access for forceps or elevators, or to section a tooth. Where this can be done with precision, it is proper to do so; but in cases where laceration of soft tissue is likely, or because of poor visibility the operator is not well oriented, it is wise to lay back a flap of soft tissue in order to expose the field.

When incisions for access are necessary, it is important that they be cut well over sound bone which is not to be disturbed, and in such a manner that adequate exposure of the operative field is obtained. Such incisions should be cut sharply and the soft tissue elevated carefully in order that this tissue, in an undamaged condition, may be accurately approximated at the completion of the operation. Careless soft tissue handling may make it possible for organisms which might otherwise be saprophytic to become pathogenic. Accurate replacement of soft tissue and careful suturing is most important to satisfactory healing. When bone is to be removed it should be done cleanly, and burnishing or burning should be avoided.

While it is true that oral tissue is remarkable for its ability to regenerate and that the operator may often deviate widely from the principles of good surgery without serious consequences, it is probable that all such deviations result in less satisfactory healing; and it is not possible to predict when such carelessness may be disastrous. It is therefore wise to review critically each operative procedure and to discover in retrospect, if possible, any detail which might have been handled more satisfactorily. To do this conscientiously will do much to eliminate errors from everyday practice.

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## FUSIFORM BACILLI AND VINCENT'S SPIRILLA IN INFECTIONS OF IMPACTED MOLARS

JOHN VERNON LENBURG, D.D.S., M.S.D.

THOMAS L. GILMER<sup>1</sup> in 1901, in lectures to the Senior Class at Northwestern University, discussed involvement of partially erupted third molars. "Many lower third molars cause trouble from the fact that they do not fully erupt. Its antagonist may be fully erupted; mastication bruises the gum and this with septic matter under the operculum causes very painful and sometimes dangerous conditions. The inflammation may subside, only to return at a subsequent time. In these cases, if I value the tooth, I consider it good practice to cut away the operculum of gum after the inflammation is under control. To control this I wash out the pus and other material contained in the pocket with pyrozone. If there is much pain, I take the crystals of muriate cocaine and grind them in 95 per cent solution of carbolic acid, and apply a small quantity of this under the operculum; this gradually relieves the pain. If it does not, I use hot water externally and if necessary, anodynes internally.

"Some texts teach removal of the second molar, thereby permitting the third molar to move forward and gain sufficient room for eruption. This may be expedient in some cases, but if it were possible I should prefer the removal of the third molar."

It was a few years later that Gilmer described a disease of the gums which he termed "acute ulcerous gingivitis," which has since been discussed very widely and under many names, such as, "trench mouth," "ulceromembranous gingivitis," and "Vincent's gingivitis." Soon after publishing a description of the disease and outlining his treatment, the bacteriologic studies made at Gilmer's suggestion by Weaver and Tuncliff established the fact that the fusiform bacillus and associated spirochetes were the active organisms. These organisms had been previously described by Plaut and Vincent in 1896 and 1898, but their presence in gingival infection had not been specifically mentioned.

Most writers on the subject of Vincent's infection of the mouth agree that (1) Vincent's infection may be found in and about the soft tissue flaps of unerupted or partially erupted teeth, (2) when Vincent's infection is present it is caused by the fusiform bacillus and Vincent's spirillum, (3) the use of oxidizing drugs in general is a desirable method of treatment.

Miller<sup>2</sup> suggests that the primary incubation zones are gingival flaps about partly erupted lower third molars. Zemsky<sup>3</sup> states that there are many ideal environments in the mouth for the growth of Vincent's organisms, such as deep gingival crevices and pockets around partly erupted teeth. Disraeli<sup>4</sup> includes tissue flaps covering unerupted teeth as areas of lowered resistance favorable to the development of Vincent's infection.

The many references to infection about the overlying gum flap of unerupted and partially erupted teeth indicates that these infections are common,

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and the frequent mention of such areas as a source for Vincent's infection suggests that most of such infections are of this type. There is very little definite information, however, regarding the incidence of Vincent's organisms in the infected gum tissues over partially erupted teeth.

#### MATERIALS AND METHODS

The present study was undertaken to determine the incidence of fusiform bacillus and Vincent's spirilla in infections about partially erupted molars under varying conditions. The material studied was smears obtained from patients in a private dental practice. This material was then prepared and studied in the Bacteriological Department of Northwestern University Dental School. The patients were unselected except that only those having one or more partially erupted third molars were used. Case history charts were kept for each patient, the age, sex, general health, occlusion, caries, restorations, condition of gingivae, past history, pain, and complaints being recorded.

The smears of one hundred teeth were studied and roentgenograms of each tooth were made. The teeth included upper and lower partially erupted teeth, some of which were acutely infected, and others with operculum not in an acute infective condition.

Case history factors were recorded and roentgenograms made; then material was obtained from the space immediately under the free margin of the operculum by means of a sterile platinum wire. Smears were made, stained, and studied microscopically.

#### DISCUSSION

In number, the lower teeth predominate (91) with practically no difference as to right or left (45-46). The difference in sex is not great, indicating that partially impacted teeth are about evenly divided between male (42) and female (58). The ages ranged from 17 to 41 years, with the average age 25.6 years.

TABLE I. BACTERIOLOGICAL FINDINGS

	NUMBER OF CASES	POSITIVE FOR VINCENT'S INFEC- TION	NEGATIVE FOR VINCENT'S INFEC- TION
Acute symptoms	35	31	4
Nonacute symptoms	65	5	60
Total	100	36	64

When this study was first begun, all partially impacted third molars which were discovered in mouths of routine patients were used. Smears and roentgenograms were made and all data were recorded. These cases, therefore, include few with severe acute symptoms. The purpose was to determine the prevalence or absence of fusiform bacilli and Vincent's spirilla about all partially erupted third molars. Of the first thirty cases studied, only one was considered to be manifesting acute symptoms, such as swelling, pain on opening, and stiffness of jaw, reddened and swollen operculum. Twenty-nine cases out of the first thirty were considered nonacute. The bacteriology was considered negative when spirochetes and fusiform bacilli did not predominate the field. Only two of the twenty-nine nonacute cases were positive bacteriologically for Vincent's infection. The one acute case was positive bacteriologically.



As the study progressed it seemed advisable to wait for acute cases which appear less frequently. Table I records the number of acute and nonacute cases, and the bacteriologic findings. Of the 35 cases with acute symptoms, 31 had a microscopic field predominated by fusiform bacilli and Vincent's spirilla, while only 4 of the 35 were negative. It is interesting to note that of the 4 negative, 2 were draining pus in large quantities from beneath the operculum.

The 35 nonacute cases included only 5 with positive bacteriology for Vincent's organisms. This was fewer than expected, since Vincent's organisms are prevalent in many mouths.

Treatment of infections in the soft tissue around partially impacted third molars is almost as varied as the number of references on the subject. These local treatments include hot applications, hot saline mouthwashes, hydrogen peroxide, counterirritants, packing of substances under the operculum, and other measures. The results of this study seem to indicate that smears should be made of each case, not only of the involved area, but of at least one other area in the mouth. If the smear indicates predominance of Vincent's organisms, the area should be treated as for ulcerative gingivitis (Vincent's infection).

In treating these cases where the smears proved positive bacteriologically, the treatment included the application of hydrogen peroxide (3 per cent), in pellets (in the office), and as a mouthwash (50 per cent) at home every two hours. The results in general were very satisfactory.

Where the presence of bacilli and spirilla have not been demonstrated, but evidence of infection is present, it is advisable to treat with a view to localization. Frequent irrigation with hot saline solution and removal of debris from beneath the operculum are beneficial. In all treatment, however, general or local resistance must be increased. Local resistance must include removal of occluding surfaces on the soft tissue over the unerupted portion of the tooth either by extraction or by grinding the occluding surface of the offending tooth of the opposing jaw.

#### CONCLUSIONS

1. There was a predominance of lower partially impacted third molars; both sexes were affected alike and with comparable incidence.
2. The condition may occur from 17 years of age, the average age occurrence being 25 years.
3. Most acute cases are positive bacteriologically in regard to spirilla and fusiform bacilli, while most nonacute cases are negative bacteriologically for these organisms.
4. All partially impacted third molars exhibiting acute symptoms should be examined bacteriologically at first examination.
5. If microscopic examination reveals predominance of fusiform bacilli and spirilla, treatment for Vincent's infection should be instituted and should include measures to increase general and local resistance.

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## COMPARISON OF SULFATHIAZOLE WITH SULFAMERAZINE IN EXTRACTION AND IMPACTION

RUSSELL G. BOOTHE, D.D.S., M.S.D.

VARIOUS sulfonamides have been used in oral surgical wounds, including sulfanilamide, sulfathiazole, and sulfadiazine. In the literature no mention seems to have been made regarding sulfamerazine in this connection.

This later drug was synthesized in 1943 by United States chemists. It has been employed by Flippin, Sifter, Comm, and Clark<sup>1</sup> in the treatment of pneumonia in 70 cases, and they compared it with sulfadiazine in 64 cases. The conclusions in these comparisons were that sulfamerazine tended to lower the temperature somewhat more rapidly than did sulfadiazine, that sulfamerazine showed higher plasma concentration of the free drug than did an equivalent amount of sulfadiazine, and that other reactions were about the same.

Davison<sup>2</sup> states that sulfamerazine is rapidly absorbed from the gastrointestinal tract and is rather slowly excreted. It seems that sulfamerazine produces fewer renal complications than sulfathiazole.

As a great deal has been written both for and against the local use of the other sulfonamides in oral surgery operation, it has appeared desirable to test clinically for the use of sulfamerazine for this purpose with regard to its effectiveness in postoperative treatment following the removal of teeth.

In the present investigation sulfathiazole was compared clinically with sulfamerazine. There were 400 cases used, 350 of routine extraction and 50 of impactions.

The cases selected were of those patients who required the extraction of teeth or the removal of impacted teeth on both sides of the arch. This made it possible to use sulfamerazine on one side and sulfathiazole on the other. The 400 cases were classified as follows: 50 of acute infection, 135 of dental caries, 96 of periodontal infection, 94 of periapical infection, and 25 of pericoronal infection.

The method of application was as follows: immediately after the operation the local area was isolated with sterile cotton rolls to exclude saliva, and the socket was dried with sterile gauze. The atomizer containing sulfathiazole or sulfamerazine was introduced into the socket and the powder dusted into the wounds. A small gauze dressing was placed immediately over the socket and the patient was instructed to bring slight pressure for a minute; it was then removed and the socket left without any other interference.

The clinical data taken on each case included the age of the patient, the sex, race, and clinical complications, such as immediate hemorrhage, postoperative bleeding, and the oral temperature. All observations were made at forty-eight and ninety-six hours after operation.

The results obtained as regards swelling and pain following extractions and application of the drugs are given in Table I.

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TABLE I

REASON FOR EXTRACTION	SULFAMERAZINE			SULFATHIAZOLE		
	CASES	SWELLING	PAIN	CASES	SWELLING	PAIN
Acute infection	25	3	5	25	3	6
Caries	68	3	2	67	3	3
Periodontal infection	48	0	1	48	0	1
Periapical infection	47	3	2	47	2	3
Pericoronal infection	12	4	0	13	3	1
Totals	200	13	10	200	11	14

It is apparent that both in the individual groups of cases and in the total cases treated with sulfamerazine and sulfathiazole, the results as regards pain and swelling are so similar for the two drugs that one could not be said to be more effective than the other. It must be borne in mind that the number of cases in which untoward symptoms appeared were too few in any event to furnish a basis for conclusive judgment.

In the group of impaction cases, the results are similar and allow of no further inference.

1. Neither sulfathiazole nor sulfamerazine showed sufficient clinical evidence for one to have any advantage over the other.

2. There were no obvious toxic reactions which resulted from the local implantation of these sulfonamides.

3. In this work there does not seem to be sufficient clinical evidence in favor of these compounds to justify their local use in oral surgical wounds; however, they may be beneficial.

4. Neither sulfathiazole nor sulfamerazine seems to relieve the pain of a dry socket.

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## A CLINICAL EVALUATION OF LOCAL ANESTHETIC SOLUTIONS OF INCREASED STRENGTH

LEO WINTER, D.D.S., M.D., AND M. L. TAINTER, M.D.

### INTRODUCTION

**D**URING the past fifty years there have appeared many publications on the clinical use of local anesthetic agents. Many of these reports have been of general or limited interest and usefulness. More recently<sup>1-3</sup> attempts have been made to devise critical methods of approach to the study of the large number of problems involved in local anesthesia and to establish the proper means for evaluation of the data obtained in these studies. There is still a crying need<sup>4</sup> for further extensive quantitative critical and objective studies of the effects of local anesthetic agents in human subjects.

It was the purpose of the present investigation, a part of which is being presented here, to obtain extensive evidence on the important effects of representative local anesthetic agents on patients. Data on two separate studies are included here. In the first study attention was concentrated on the onset, duration, etc., while in the second study the patient reactions were the primary objective. The data included in Tables I to III are from the first study while those of Table IV are from the second. In this presentation an attempt will be made to dispel uncertainties about the absolute and relative tolerance of some commonly used anesthetic solutions.

### STATEMENT OF PROBLEM

Using the blind test technique on a large group of patients well distributed as to age, sex, and physical condition, we set out to study representative local anesthetic agents from the standpoint of the following: volume of solution required, onset time of anesthesia, duration of anesthesia, blood pressure changes, pulse rate changes, grade of anesthesia, amount of bleeding, and patient reactions. In this work we have been guided by the methods employed by Tainter, Throndson, and Moose.<sup>1, 3</sup>

Although studies have been initiated on some thirteen solutions, the present report is concerned only with the following four isotonic solutions:

Novocain 2 per cent with Epinephrine 1:50,000

Novocain 2 per cent with Cobefrin 1:10,000

Novocain 3 per cent with Epinephrine 1:50,000

Novocain 2 per cent, Pontocaine 0.15 per cent, Cobefrin 1:10,000

Each was made isotonic by the addition of the appropriate amount of sodium chloride. As data on the other solutions become available, they will be presented in future publications.

From the Department of Oral Surgery, College of Dentistry, New York University, and Bellevue Hospital, New York, N. Y.

This investigation was made possible by a grant made by Cook-Waite Laboratories to New York University.

## PROCEDURE

The solutions used in the clinic were provided in such form that the clinicians were at all times unaware of the identity of the products under test. The solutions were put up in cartridges in lots of fifty to each of twenty containers. Each container carried its own key number so that the observer could not know what formula he was using at any time. Furthermore, the solutions were frequently interchanged, alternating from one to another in an irregular manner, so that such factors as change in personnel and variation in patients observed were equalized as well as possible. This so-called blind test technique is absolutely necessary in clinical studies of this type if one is to arrive at reliable data which are not colored by subjective influences.

All injections were made by senior dental students or instructors. At the time of injection the quantity of solution used was recorded and any objective symptoms were noted. All blood pressure readings and pulse rates were taken by a registered nurse before and one minute after injection. It was thought that since dentists have to recognize and cope with reactions, when they occurred in this four-solution clinical investigation they should be evaluated by a dentist. In the second series, recorded in Table IV, all observations were made by one person, a dentist, who devoted his time exclusively to interpreting and recording manifestations of patient reactions and time of onset and duration of anesthesia. The dentist, like all operators concerned in this study, was unaware of the identity of the solutions being injected. The data sheets were assembled and statistically analyzed by different persons from those making the observations, so that subjective influences from this source were also rigidly excluded.

In all cases the criterion used in determination of onset time was the presence of sufficient anesthesia for the performance of the operations. Duration was judged by recurrence of sensation as determined by the use of an explorer. The degree of bleeding was recorded by each observer in accordance with standards previously agreed upon.

## LOCAL ANESTHESIA

Before an evaluation of an anesthetic solution can be made, it is necessary not only to evaluate and standardize the solutions, the patients, methods of recording and procedures entailed, but the technical processes involved in administering the anesthetic are also of paramount importance. Local anesthesia is dependent upon knowledge of the regional anatomy involved. With this in mind, a complete anatomic study of the head and neck was made prior to the survey. The basic anatomic considerations are illustrated in Plates I and II, and Figs. 1, 2, 3, 4, and 5.<sup>10</sup>

## TECHNIQUE OF INJECTIONS

The technique of these procedures is all-important in that comparable results will be achieved only if the procedure of the administration of the local anesthetic is completely systematized. Local anesthesia implies more than the administration of intraoral novocain anesthesia; it includes, as well, extraoral block and infiltration methods. Therefore, on the basis of the anatomic dissections and the procedures that have been employed in the past with success, the following techniques of injection anesthesia were employed.

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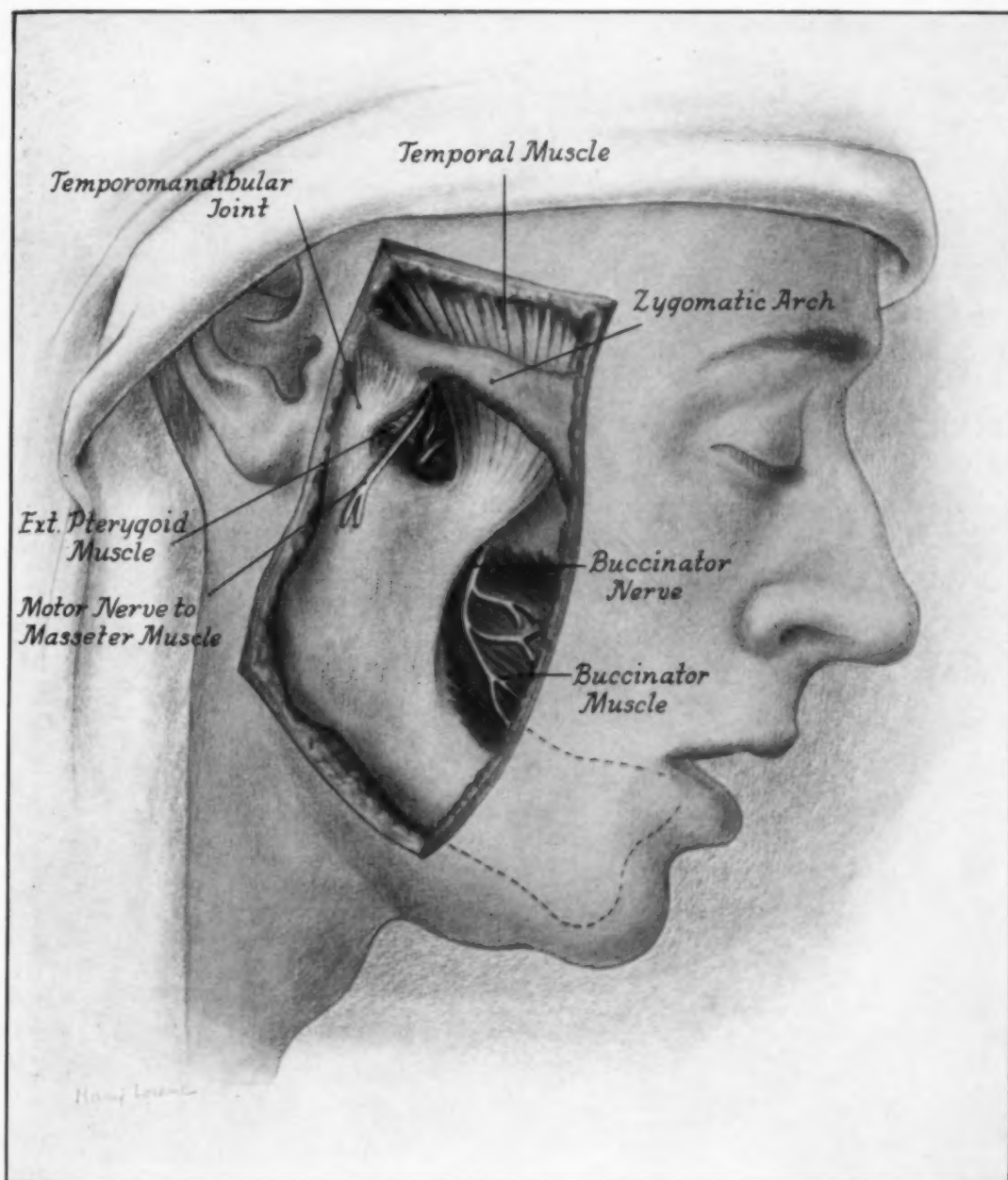


Plate I.—Deep dissection to lateral aspect of mandible wherein muscle and parotid gland have been removed.

(Plates I to V from Winter: Textbook of Exodontia, The C. V. Mosby Co.)



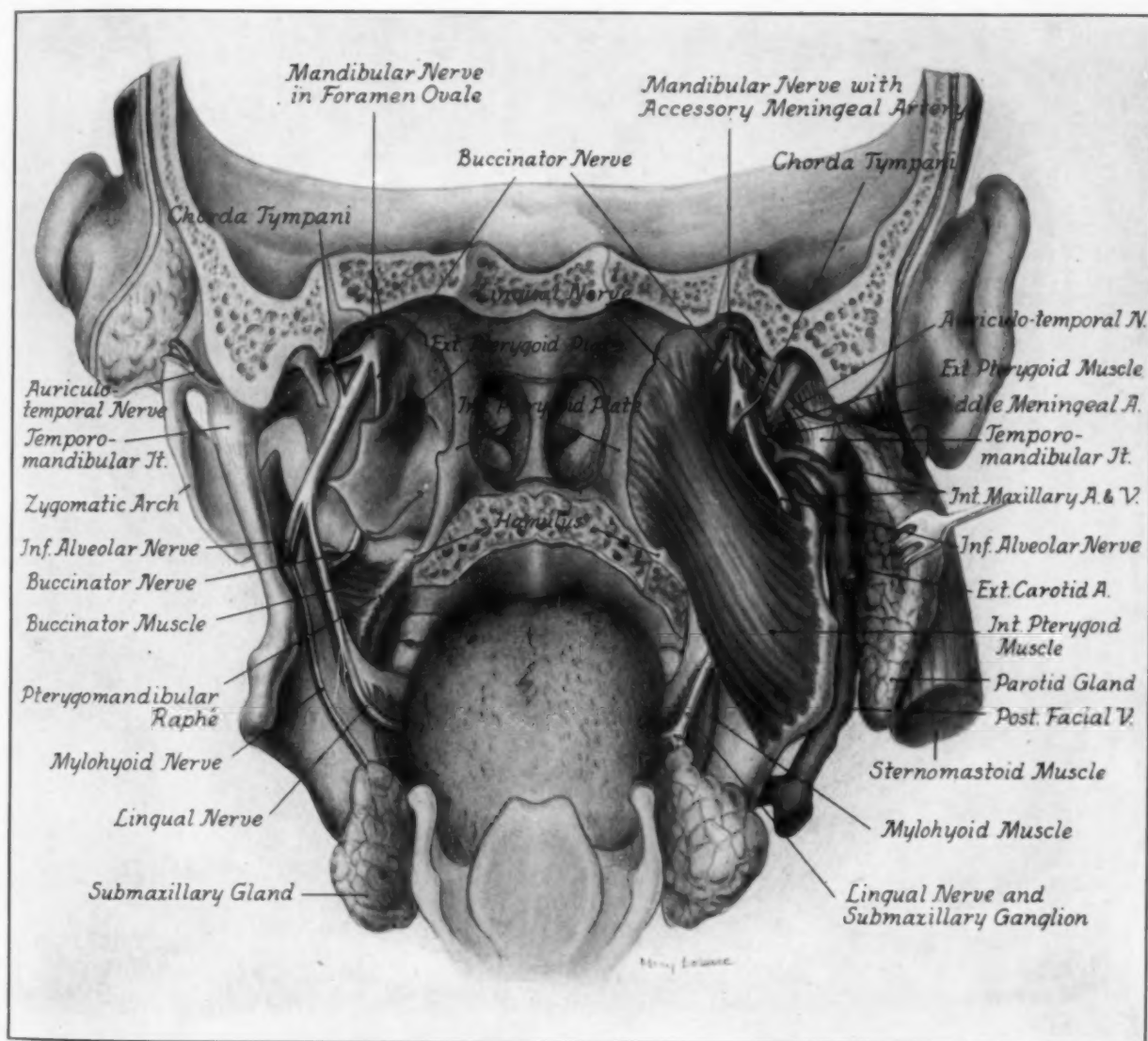
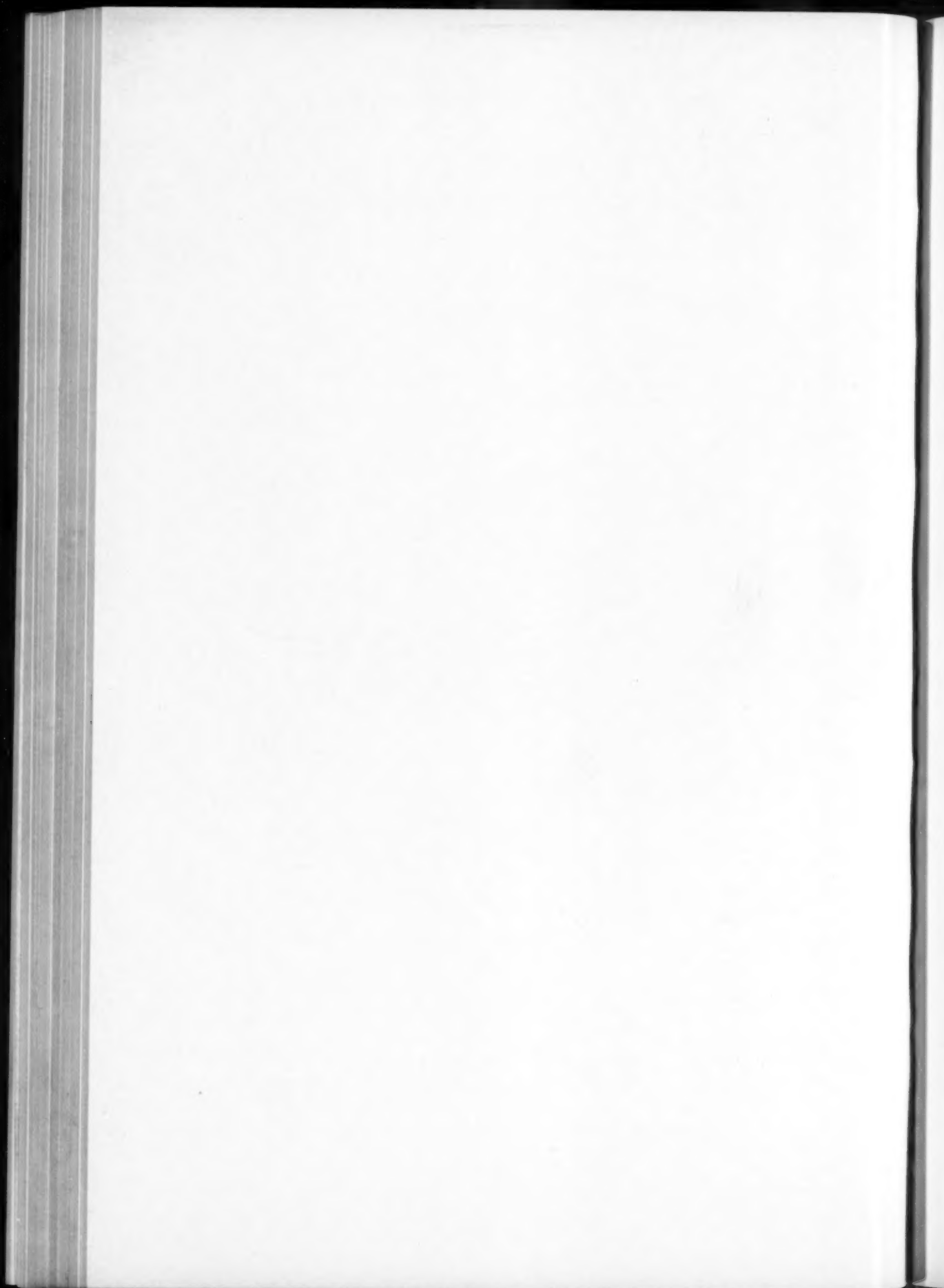


Plate II.—Posterior view of the topography of the mandibular nerve. The left side shows the relation of the mandibular nerve to the skeleton. The right side shows the relation of the mandibular nerve to the pterygoid muscles.





*Inferior Alveolar.*—Blocking the inferior alveolar nerve at the lingula, on the inner border of the ramus, will secure anesthesia of that particular side of the mandible up to the lateral incisor. The technique employed is as follows:

The patient's head is in a vertical position and the mouth open to the fullest extent. With the body of the mandible parallel to the floor, the index finger is placed posterior to the third molar where the external oblique ridge

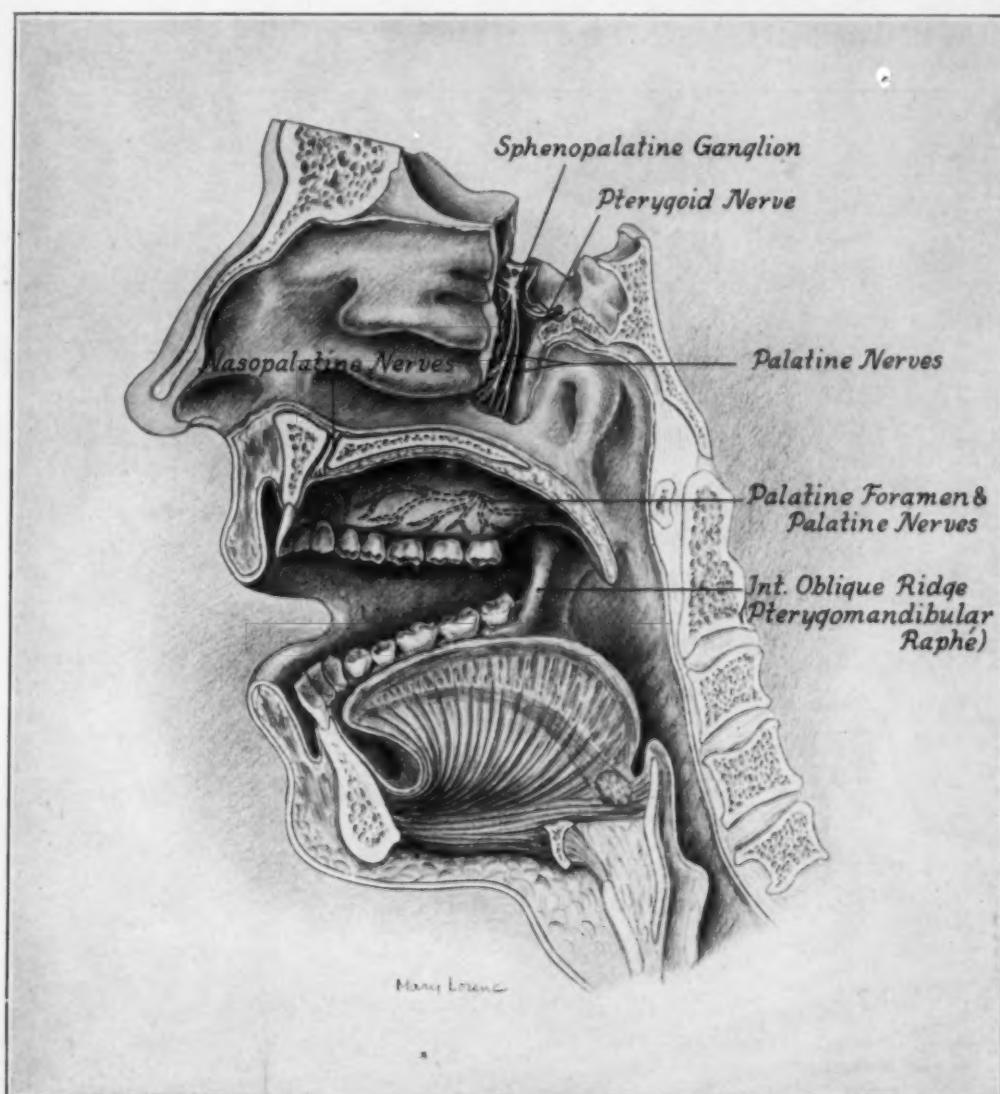


Fig. 1.—Sagittal section through maxilla, mandible, and sinuses, right side.  
(Fig. 1 to 9 from Winter: *Textbook of Exodontia*, The C. V. Mosby Co.)

can be palpated. Directly lingual to this ridge lies the retromolar fossa. The index finger is placed inside the retromolar fossa, and the fingernail automatically touches the internal oblique ridge. The finger is kept in this position, sterile gauze is carried by means of thumb forceps to the point where the fingernail rests, and the mucous membrane lingually is dried. Tincture of aconite and iodine is then applied to the area, fixing the surface bacteria.

The syringe, with the bevel edge of the needle toward the bone, is brought in from the opposite side in the area of the premolars and is made to penetrate the mucous membrane at a point approximately bisecting the fingernail. The needle is passed along the inner border of the ascending ramus until the resistance of the bone to the needle is lost. Thereafter the needle is passed beyond the lingula, the sharp prominence which guards the orifice of the inferior alveolar canal, and the solution is deposited by aid of a slow, pumping motion. The anatomic relationships are illustrated in Fig. 6, and the technique of the injection in Figs. 7 and 8.<sup>10</sup>

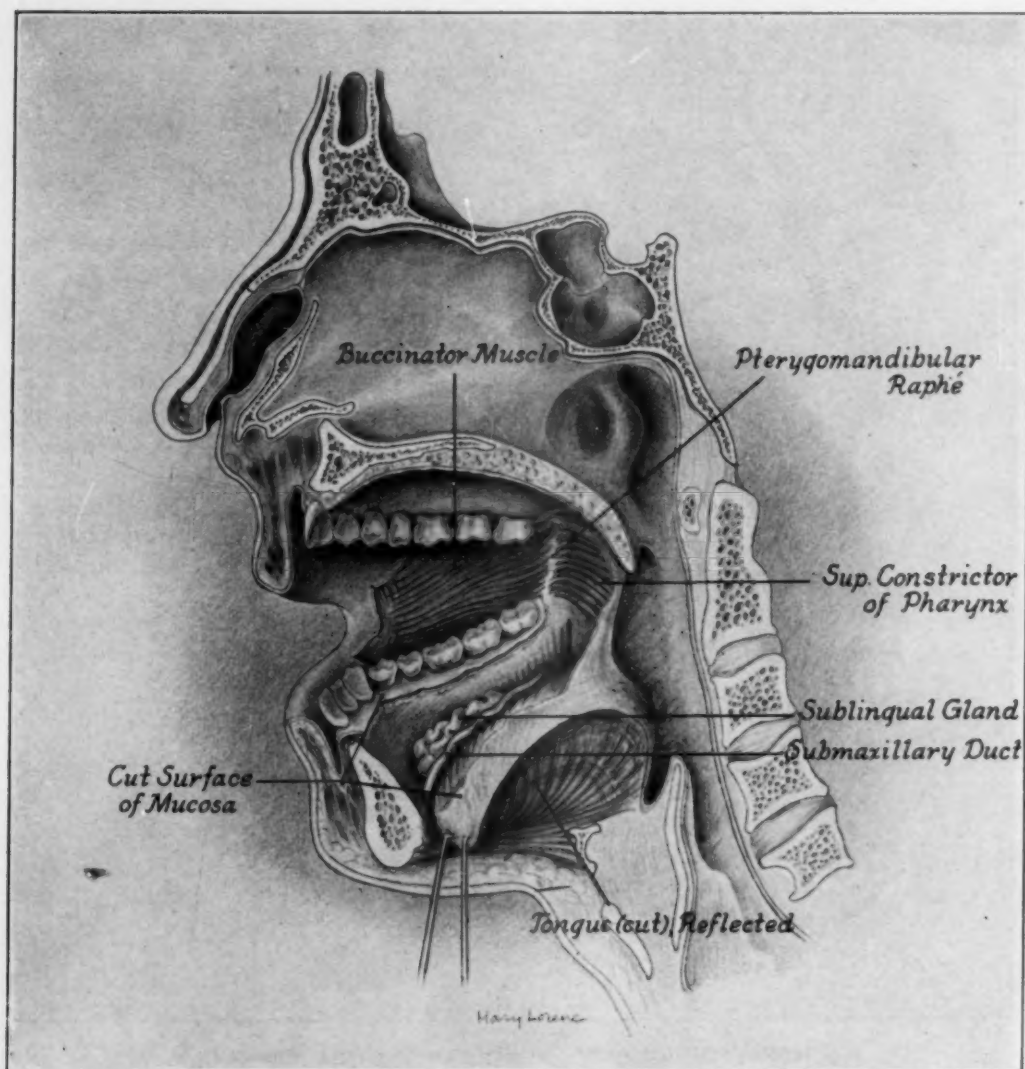


Fig. 2.—Sagittal section revealing dissection of the structures with the mucous membrane at the pterygomandibular raphe lifted.

**Posterior Superior Alveolar Injection.**—The anesthetization of the posterior superior alveolar nerve, which supplies the three maxillary molar teeth with the exception of the lingual mucosa and the mesial buccal root of the first molar (which may be supplied by the middle superior alveolar nerve), is accom-

plished by the following technique: Index finger of the left hand is placed into the vestibule of the mouth under the zygomatic process with the mouth half open. The corpus adiposum buccae is drawn out of the field of operation by the natural lateral displacement of the index finger. The syringe is held in a pen grip with the point of insertion approximating the apex of the distal

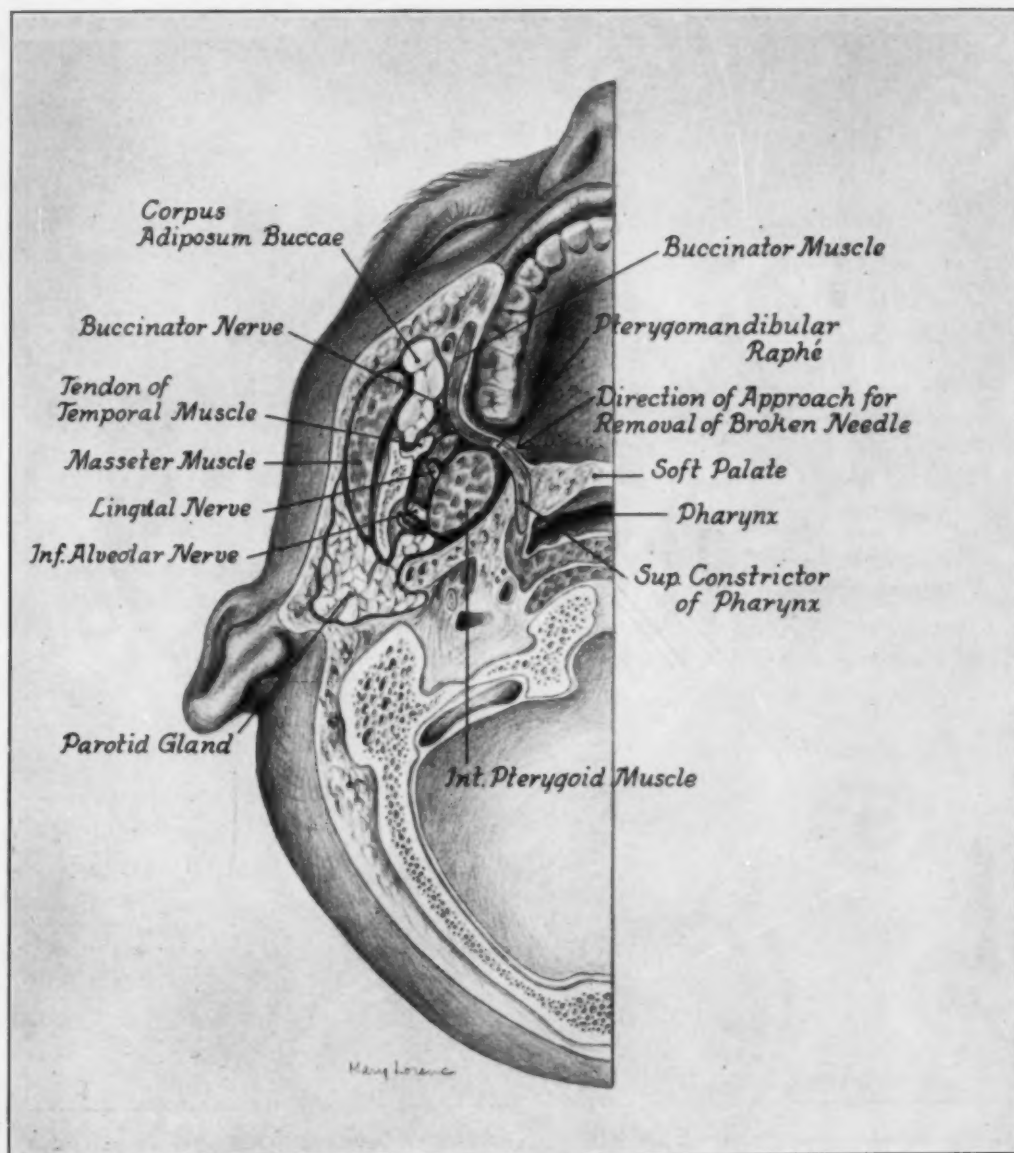


Fig. 3.—Horizontal section through the face at the level of the angle of the mouth to show the route which must be taken for the removal of a broken needle.

root of the next to the last molar in position. The needle is then inserted upward, backward, and inward, at all times hugging the alveolar process. Fluid is deposited with a slow, pumping motion. It is highly vital that the mouth be kept in a half-open position so as to allow for the complete retraction of the corpus buccae, for if there is failure to accomplish this adequately,

there is the possibility that the injection will be made into the fatty mass with the resultant balloonlike swelling.

*Infraorbital Injection.*—Infraorbital injection may be employed for the anesthetization of the area of the first and second premolars, cuspids, lateral and central incisors of the particular side involved. The index finger of the left hand is used to palpate the supraorbital notch, and in a direct line with

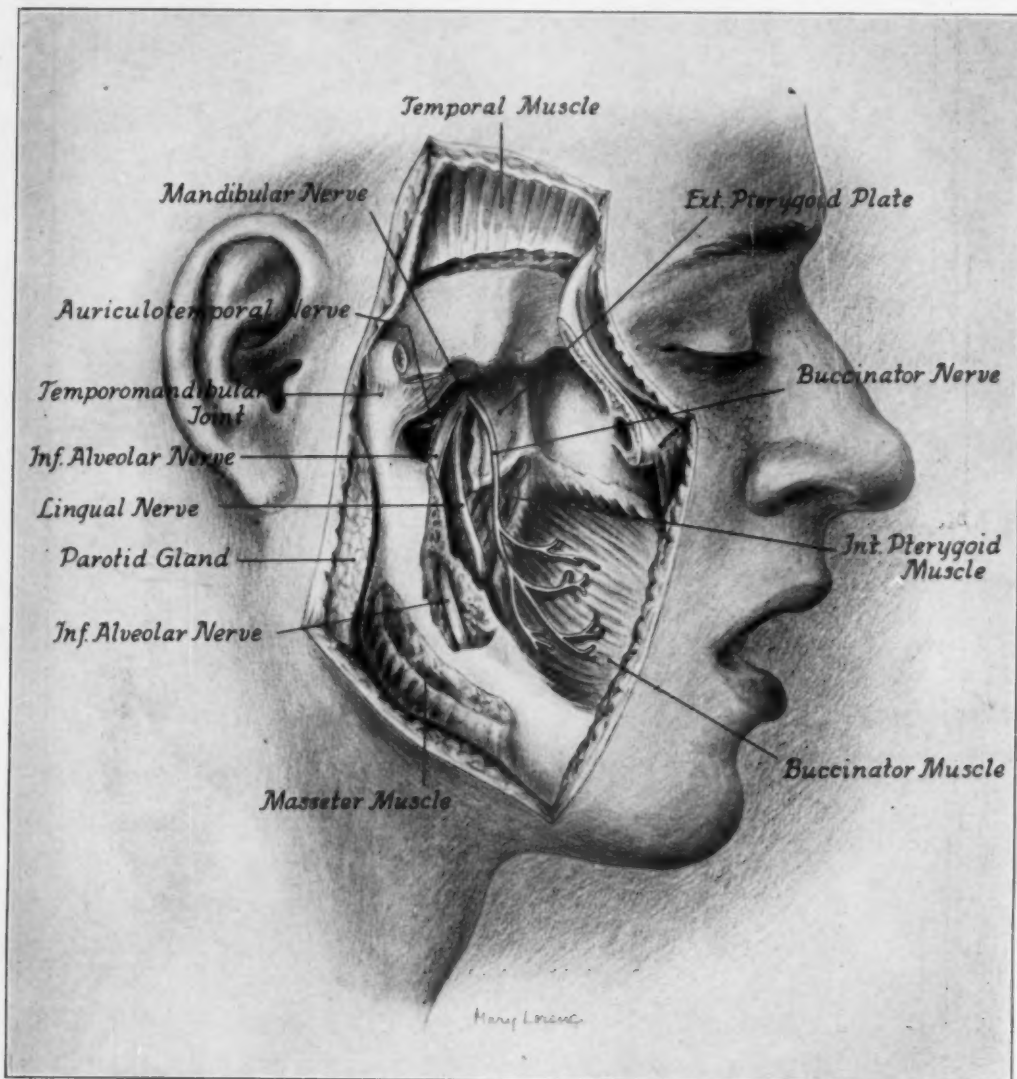


Fig. 4.—Infratemporal fossa. Zygomatic arch, masseter, temporal and external pterygoid muscles removed; inferior alveolar canal opened.

this notch below the infraorbital ridge, which can be palpated, is the infraorbital foramen. The thumb of the same hand is placed below the infraorbital ridge in a direct line with the supraorbital notch. The ball of the thumb will then be resting over the infraorbital foramen. The lip is everted with the index finger. The syringe is held in a pen grip, the bevel edge of the needle toward the bone, and the point of the needle is inserted in the reflection



of the mucous membrane above the apex of the second premolar. The syringe and needle are directed upward and parallel to the long axis of the secondary premolar. The solution is deposited slowly and will be felt beneath the palpating finger. This direction of the needle will avoid the canine fossa and

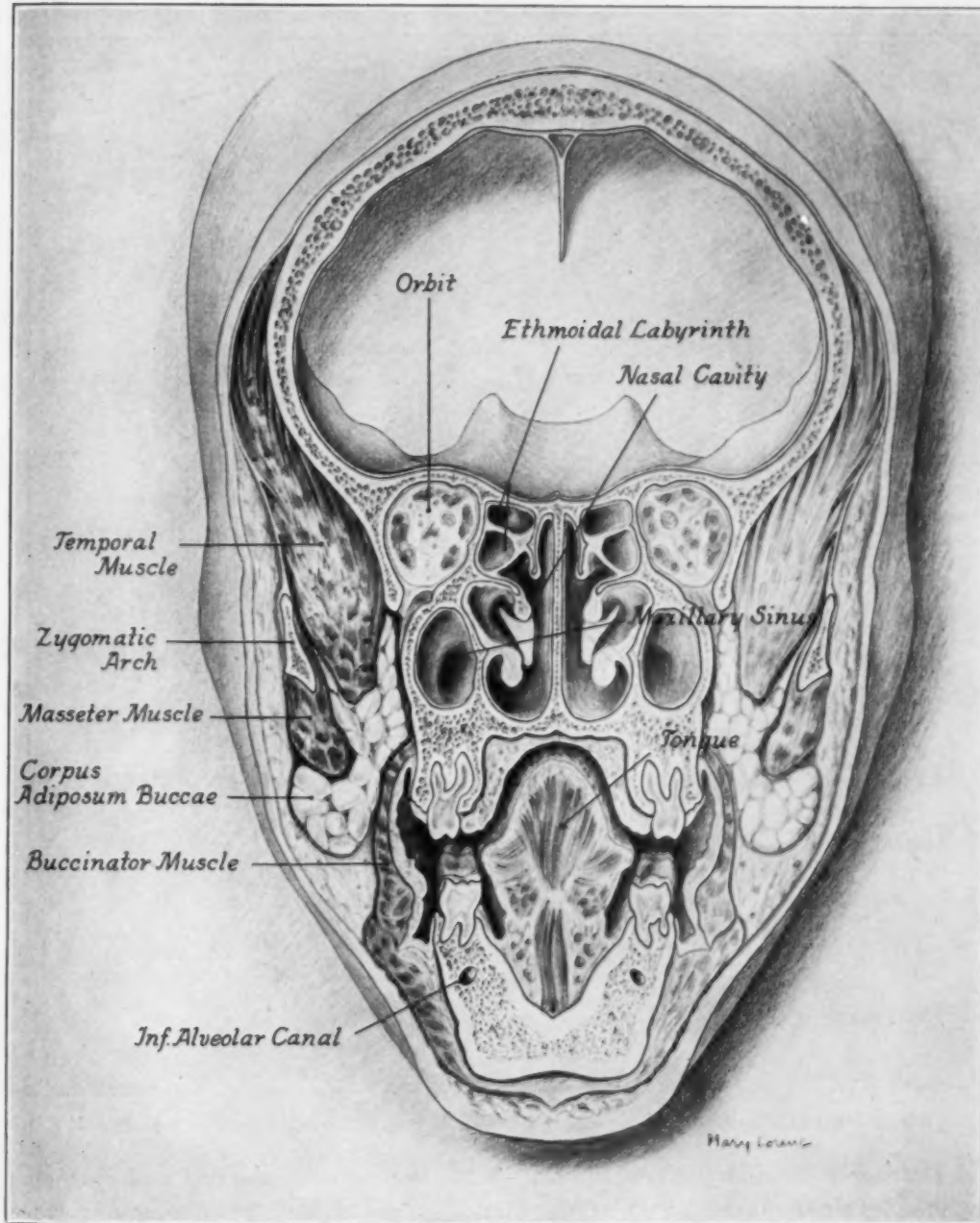


Fig. 5.—Section of head through molar region.

will pass through adipose tissue between the quadratus labii superioris and the caninus muscles. Avoiding the penetration of either muscle will prevent ecchymosis. Light massage of the face in this region often aids in hastening the anesthetic effect.

*Infiltration.*—The technique employed consists of drying and fixing the surface bacteria. The needle, with the bevel directed toward the bone, is inserted at a point midway between the gingival margin and the apex of the tooth. The solution is deposited with a slow, pumping motion.

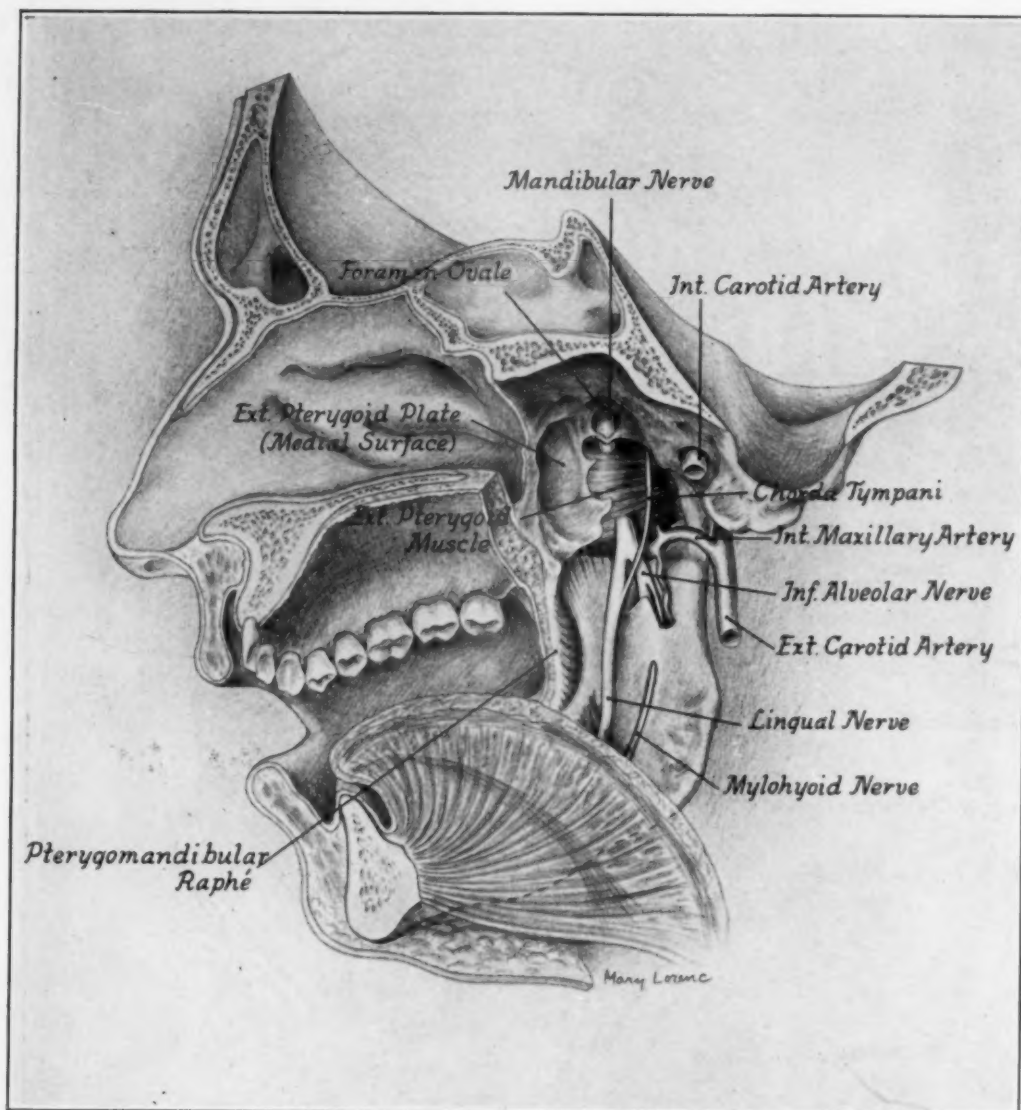


Fig. 6.—Lingual and inferior alveolar nerves piercing external pterygoid muscle.

*Extraoral Maxillary Nerve Block.*—With the patient opening and closing the mouth, the sigmoid notch of the mandible is outlined by placing the tip of the index finger of the left hand just beneath the zygomatic arch; the depression thus palpated is the notch; the bisection of the tip of the index finger marks the middle point of the zygoma. Under infiltration, subdermal anesthesia, a wheal is created in the area of the sigmoid notch just beneath the skin surface. After skin anesthesia has been established, the index finger is placed at a point denoting the sigmoid notch, and a needle (80 mm. long—

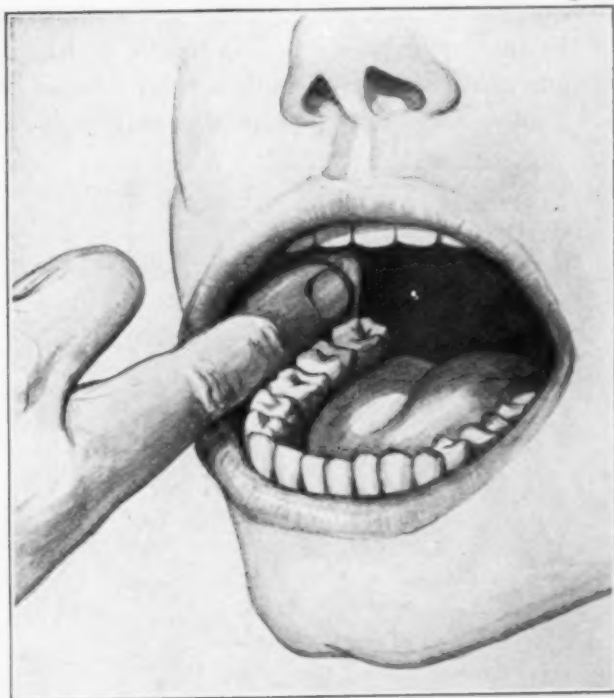


Fig. 7.—The ball of the finger resting in the retromolar fossa.

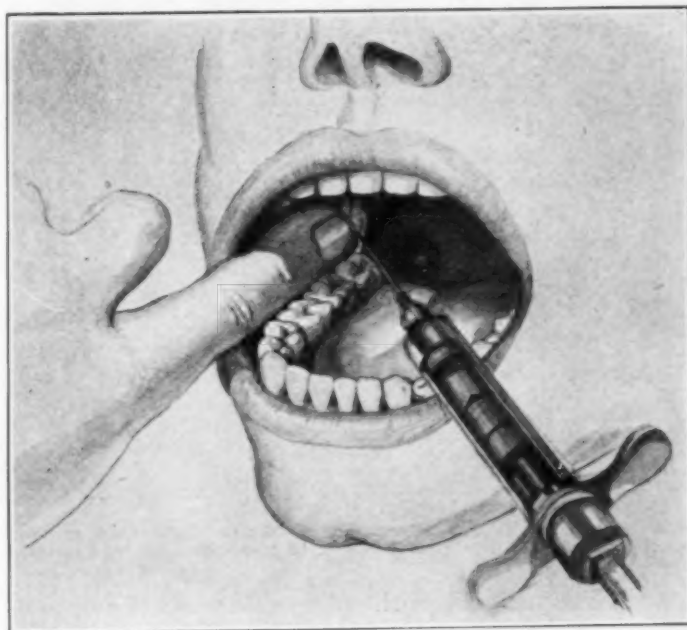


Fig. 8.—Point of penetration of the needle into the mucous membrane for the inferior alveolar injection.

Labat) is passed through the point bisecting the fingernail and inserted until contact with the bone has been made. At this point the needle is in contact with the base of the pterygoid process. The needle is drawn out sufficiently to allow for a change of direction and is then reintroduced in a forward and upward direction to the same depth as it was originally introduced. The

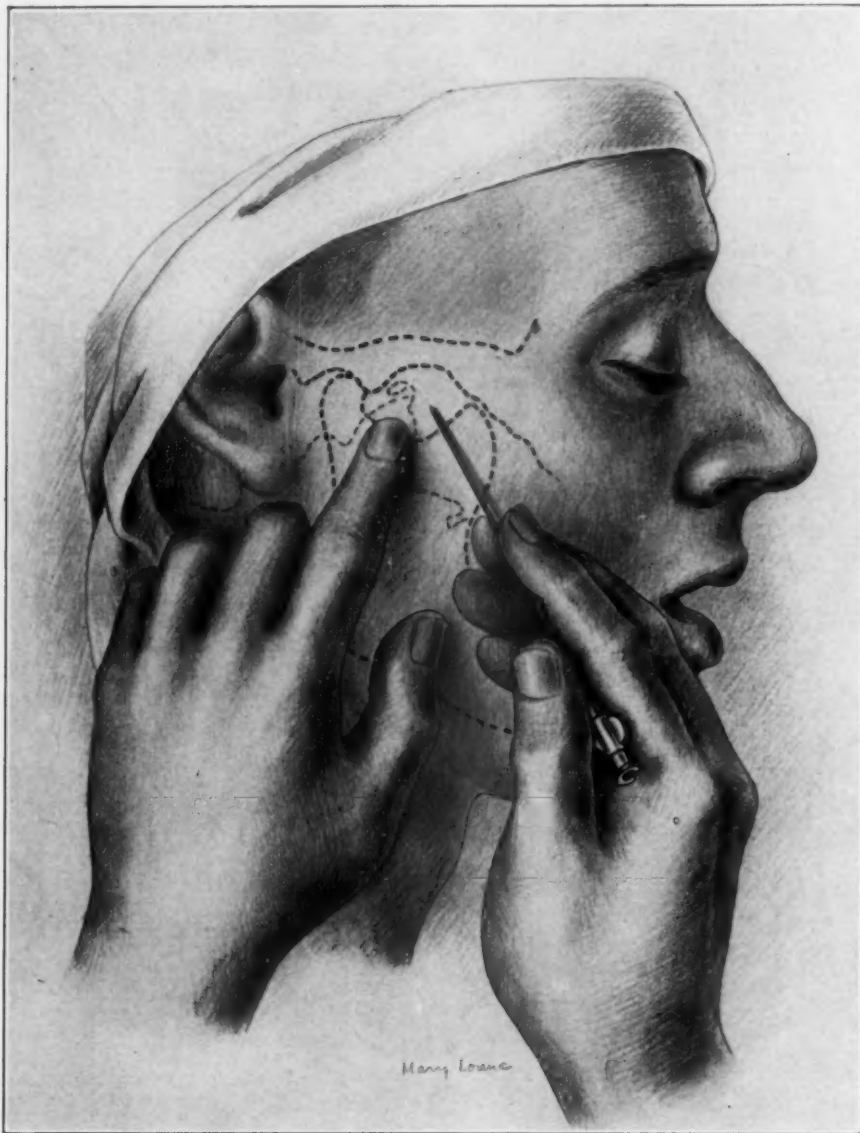


Fig. 9.—Mandibular nerve block. Index finger of left hand palpating for inferior border of sigmoid notch with Labat needle in position to puncture skin at bisection of nail.

needle has now entered in front of the pterygoid process and is in the pterygo-palatine fossa. The syringe with the anesthetic solution is connected, aspiration test is performed, and, if negative, the solution is slowly deposited. This produces complete anesthesia of the maxillary division of the trigeminal nerve. The anatomy of this area and the routes of injection are illustrated in Plates III, IV, and V and Fig. 9.<sup>10</sup>



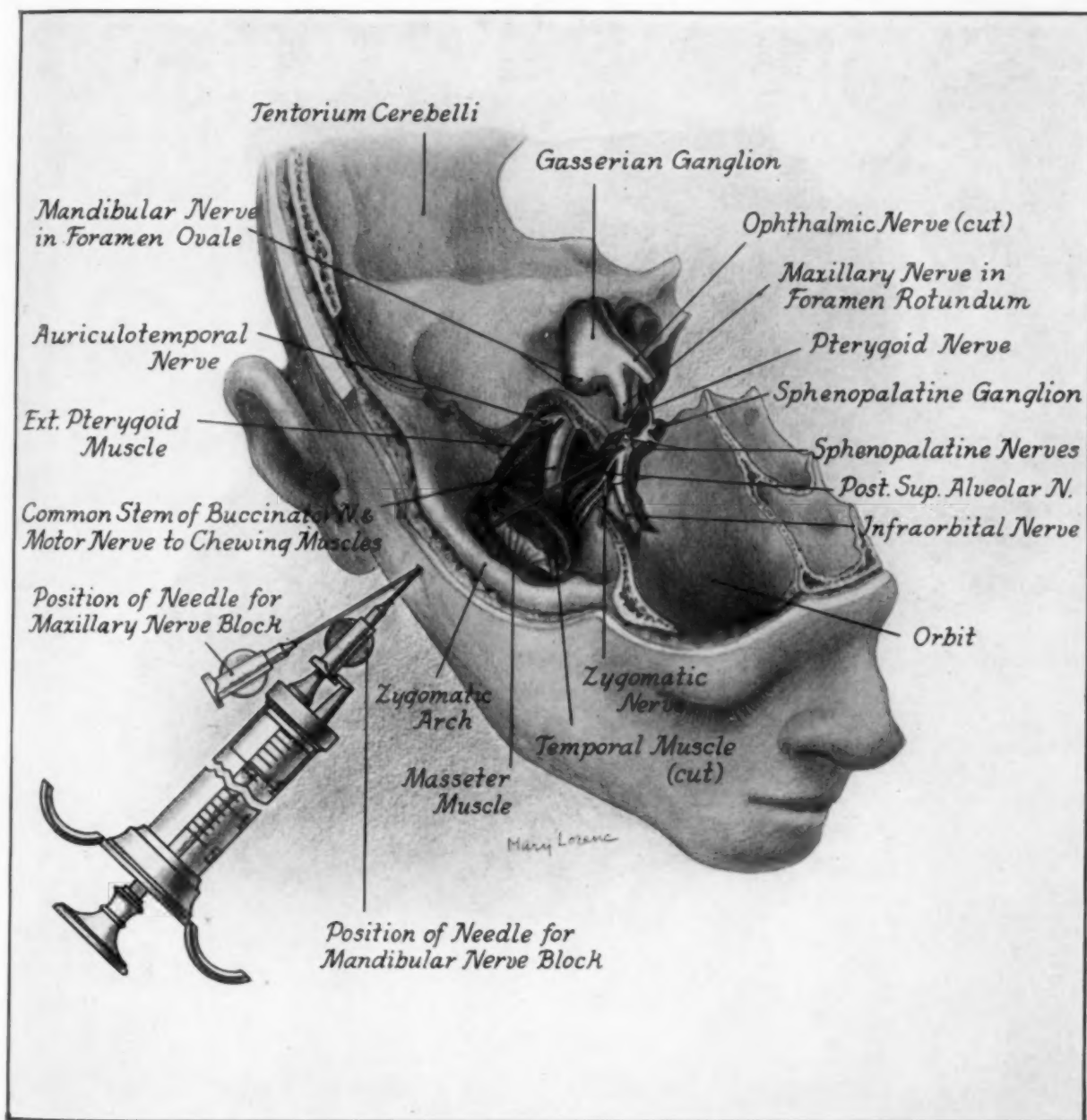


Plate III.—Infratemporal fossa viewed from above. Blue needle directed to the maxillary nerve in the pterygopalatine fossa. Black needle directed to the mandibular nerve after its passage through the foramen ovale.



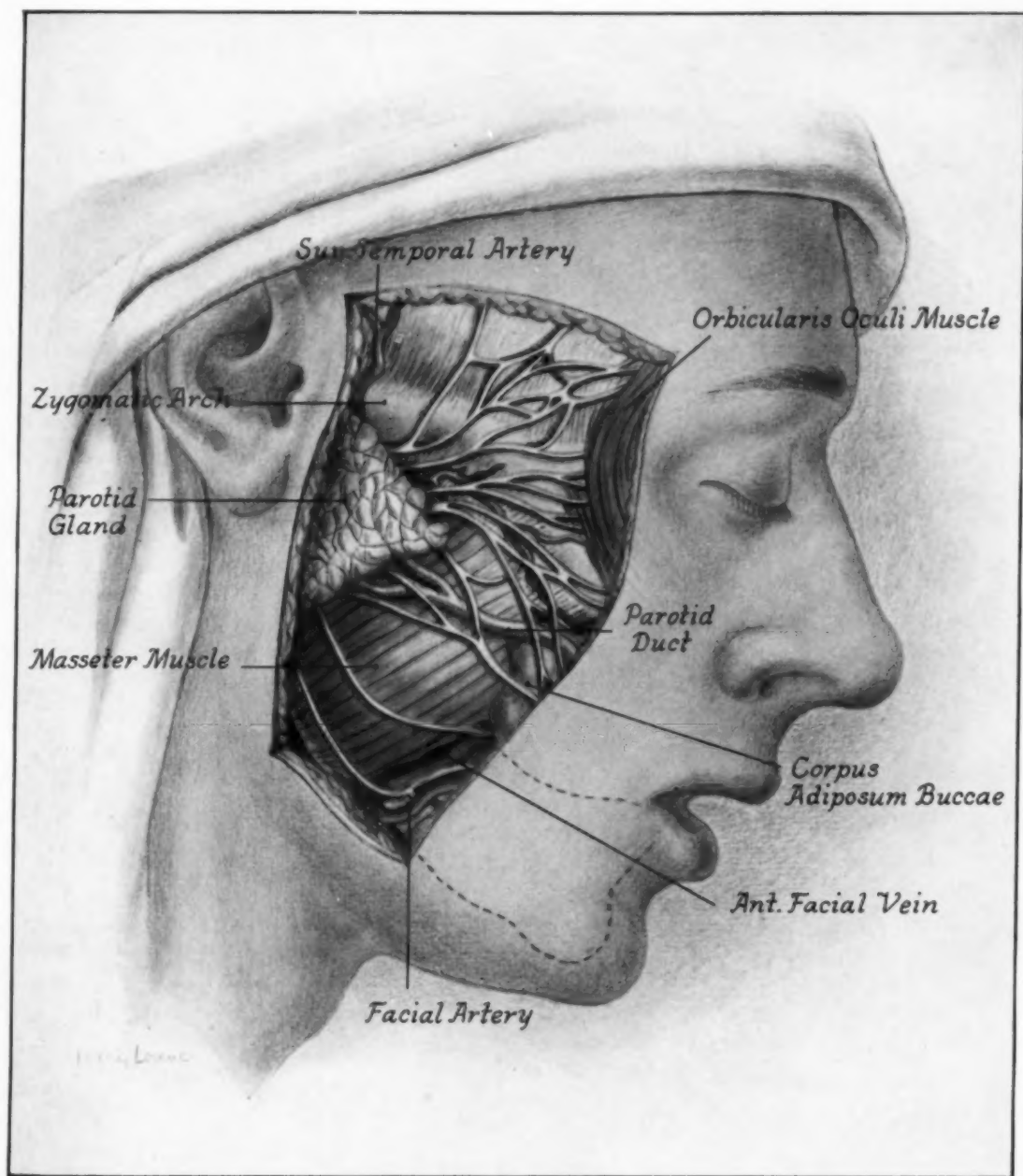


Plate IV.—Superficial lateral region of the face showing the profuse anastomoses of the branches of the facial nerve.





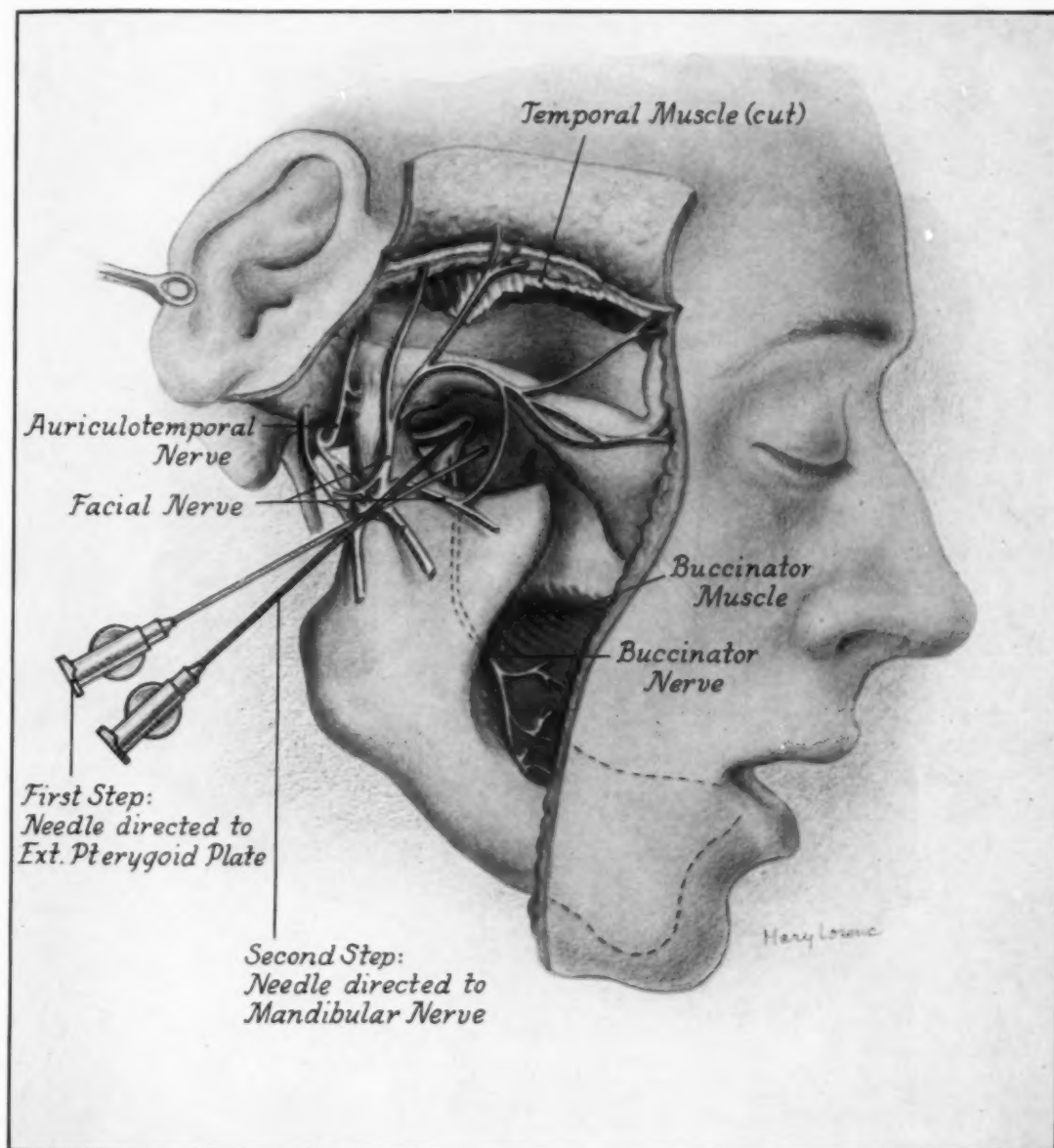


Plate V.—Position of needle in contact with external pterygoid plate. Needle removed slightly and direction changed posteriorly to foramen ovale.



*Extraoral Mandibular Nerve Block.*—The method employed for the blocking of the mandibular division of the trigeminal nerve is similar to that for the maxillary nerve with the following deviation in technique: Instead of passing the needle in front of the pterygoid process, the needle is directed backward to the foramen ovale, which lies just posterior to the base and is at about the same depth as the outer surface of the lateral plate of the pterygoid process. The setting of the recorder at a distance of 1 cm. from the surface of the skin in this situation is to prevent the introduction of the needle into deeper structures where damage may be done. Paresthesias in the lower jaw are the best indications that the nerve has been reached.

## EXPERIMENTAL PROCEDURE AND RESULTS

The results of our investigations on 2,382 patients injected according to the above techniques are recorded and summarized in the tables of data. For each value obtained in Tables II, III, and IV, the familiar standard error of the statistician is given. This indicates the extent of the variability of the average values. The procedure involved in arriving at these values is available in any standard textbook on statistics, and therefore need not be presented here. Suffice it to say that it is a measure of the range within which the average value obtained might fluctuate from chance variation. To be more specific, there is a two to one chance that there will not be a variation from the average value in either direction of more than one standard error if the entire study were to be conducted a second time. The percentage probability has also been included in Tables II, III, and IV. This gives quantitative expression to the probability that differences between the solutions indicated are or are not due to chance alone. If the probability value is equal to or less than 0.05, the difference between two solutions compared is considered statistically significant.

Particular attention should be paid to the fact that we have used Novocain\* 2 per cent solution with epinephrine 1:50,000 as the standard against which each of the other three solutions is compared. This combination was selected as the reference solution because all dentists are familiar with its properties and it has been used for this purpose in previous publications.

TABLE I. SUMMARY OF ANESTHETIC SOLUTIONS EMPLOYED, NUMBER OF CASES PER SOLUTION, AND TYPES OF INJECTION IN FIRST SERIES

SOLUTION	NUMBER OF CASES	TYPE OF INJECTION							
		INFILTRA-TION		INFERIOR ALVEOLAR		POSTERIOR SUPERIOR ALVEOLAR		INFRA-ORBITAL	
		NO.	%	NO.	%	NO.	%	NO.	%
Novocain 2% with epinephrine 1:50,000	330	171	52	131	40	23	7	5	2.0
Novocain 2% with cobefrin 1:10,000	326	156	48	139	43	28	8	3	1.0
Novocain 3% with epinephrine 1:50,000	543	289	53	215	40	37	7	2	0.4
Novocain 2%, pontocaine 0.15%, cobefrin 1:10,000	561	279	50	252	45	29	5	1	0.2
Totals	1,760	895	51	737	42	117	7	11	0.6

\*Novocain is the Winthrop Chemical Company, Inc., brand of procaine hydrochloride U. S. P.

In Table I are summarized by solutions the types of injections used in the 1,760 patients in the first series. In Table IV is given the same information on the 622 patients of the second series. As can be seen in these tables, the number of cases studied was sufficiently large to make statistical analysis reliable. It will be noted that from solution to solution there was achieved an equal distribution of cases according to type of injection. This is further evidence of the objectivity and effectiveness of the "random sampling" methods used in this study. Since it is obviously impossible to select matched groups, the effects due to individual variation may be cancelled out by using a large number of patients. The elements of the past history, age, sex, and any physical disabilities were recorded and have been analyzed and reported separately.<sup>8</sup> Those which significantly affected the response to the local anesthetic were equally distributed between the solutions under test by the method of randomization employed, and hence did not distort the average values. All of the patients presented themselves for dental treatment at New York University School of Dentistry and Bellevue Hospital.

TABLE II. COMPARISON OF RESULTS OF THE FOUR SOLUTIONS IN FIRST SERIES OF 1,760 PATIENTS WITH RESPECT TO VOLUME INJECTED, ONSET TIME, DURATION OF ANESTHESIA, AND CIRCULATORY CHANGES

SOLUTION	VOLUME RE- QUIRED (C.C.)	ONSET TIME OF ANES- THESIA (MIN.)	DURATION OF ANESTHESIA (MIN.)	BLOOD PRESSURE CHANGES IN MM. HG		PULSE RATE CHANGES PER MIN.
				SYSTOLIC	DIASTOLIC	
Novocain 2% with epinephrine 1:50,000	3.4 ± 0.03	3.1 ± 0.17	190.3 ± 20.26	5.3 ± 2.28	-3.1 ± 3.71	8.3 ± 1.19
Novocain 2% with cobefrin 1:10,000	3.3 ± 0.10	3.3 ± 0.15	175.0 ± 27.29	9.0 ± 2.32	3.0 ± 1.09	-0.2 ± 1.21
Probability of no difference from novocain 2% with epinephrine 1:50,000	.34	.38	.66	.25	.12	<.001
Novocain 3% with epinephrine 1:50,000	2.8 ± 0.07	3.3 ± 0.34	136.8 ± 10.88	4.6 ± 1.60	-2.9 ± 1.08	7.8 ± 0.90
Probability of no difference from novocain 2% with epinephrine 1:50,000	<.001	.60	.02	.80	>.90	.74
Novocain 2%, pontocaine 0.15%, cobefrin 1:10,000	2.9 ± 0.06	3.5 ± 0.13	215.0 ± 15.00	6.8 ± 2.69	2.6 ± 1.04	-9.2 ± 2.37
Probability of no difference from novocain 2% with epinephrine 1:50,000	<.001	.06	.34	.67	.14	<.001

In Table II are given the volumes of solution required for anesthesia in the first series. This should be compared with the same data on the patients in the second series, set forth in Table IV. It will be noted that the average values for each series are practically identical. This gives added confidence in the comparability of the two sets of data derived from the two series of observations.

The volumes of solution required for novocain 2 per cent with epinephrine or Cobefrin\* as the vasoconstrictor were practically identical. However, when 0.15 per cent Pontocaine\* was added to the novocain-cobefrin solution the

\*Cobefrin is the Winthrop Chemical Company, Inc., name for racemic 3,4 dihydroxy-phenylpropanolamine hydrochloride. Pontocaine is the brand of tetracaine hydrochloride U. S. P. marketed by the Winthrop Chemical Company, Inc.



volume of solution needed was sharply reduced by 0.4 to 0.5 c.c. or about 14 per cent. The same sized reduction of the volume of anesthetic used was secured by raising the novocain concentration to 3 per cent, a 50 per cent increase in the amount of the anesthetic. These differences in volumes used are statistically significant. From the practical standpoint these figures are of interest, since at least a part of any added toxicity inherent in the stronger anesthetic solutions would be compensated for by the smaller volume of solution required.

As might have been anticipated, the differences in the onset time of anesthesia for the four solutions are insignificant (Table II). Each would be satisfactory when judged from this standpoint alone.

The findings on duration of anesthesia summarized in Table II should be considered in light of conditions prevailing at the clinic, as well as the requirements for anesthesia in the operation performed. In all cases anesthesia was of sufficient average duration. The finding that the duration of anesthesia with 3 per cent novocain solution is much less than that for 2 per cent solution may be related in part to the smaller volume used of the more concentrated solution but probably is due to the vasodilator action of the novocain, which in the higher concentration more effectively overcomes the vasoconstriction of the epinephrine and thus shortens the anesthesia.

The longest duration of anesthesia occurred with novocain-pontocaine-cobefrin, which anesthetized twenty-five minutes longer than did the 2 per cent novocain-epinephrine solution, and forty minutes longer than the one with cobefrin. However, these differences are not fully significant statistically. There is no doubt that the seventy-eight-minute greater duration of the novocain-pontocaine-cobefrin over the 3 per cent novocain-epinephrine is significant and important, particularly in view of the equally small volumes of each injected. It must be pointed out, however, that the clinical experiments performed do not provide a completely satisfactory basis for comparing duration of anesthesia in these solutions, since the patients cannot always be kept under observation until the anesthesia is gone. For this purpose the use of prolonged operative procedures would give more reliable *absolute* values. The recognition of this restriction on the interpretation of the *absolute* values observed does not, however, deny their *relative* value in comparing solutions run in parallel under the identical conditions. It is obvious, therefore, that the pontocaine-containing solution gives a longer-lasting anesthesia than do the others, which difference would doubtless be even more striking if identical volumes had been used for each.

The blood pressure changes for the four solutions are small. There appear to be no significant differences between the solutions in this regard, except that the cobefrin produced a minimal rise in diastolic pressure as well as in systolic, whereas the epinephrine solutions produced a slight drop in diastolic pressure with a concomitant rise in systolic, thereby increasing the pulse pressure. This difference in the action of cobefrin and epinephrine was pointed out by Miller and Stuart.<sup>9</sup> It will be noted that there are differences in pulse rate changes produced by the four solutions. These differences, while not extreme, appear to be significant. Actually the two solutions containing cobefrin allowed a slight decrease in the pulse rate, whereas the solutions con-

taining epinephrine produced an increase in pulse rate. These findings are comparable to those of Tainter, Thronson, and Moose.<sup>2</sup> Undoubtedly there will be an increase over the normal pulse rate when excitable individuals present for dental service. The pulse rate can be expected to return to normal following an injection, unless prevented by the stimulating action of the injected vasoconstrictor. Miller and Stuart<sup>9</sup> demonstrated that there was a greater increase following the injection of novocain solutions containing 1:20,000 epinephrine than with either epinephrine 1:50,000 or cobefrin 1:10,000 and that the pulse rate returned to normal sooner following the injection of 1:10,000 cobefrin with novocain 2 per cent than with either of the solutions containing epinephrine.

TABLE III. COMPARISON OF RESPONSE TO LOCAL ANESTHETIC INJECTION OF THE 1,760 PATIENTS OF THE FIRST SERIES IN RESPECT TO DEGREE OF ANESTHESIA, AMOUNT OF BLEEDING, AND ATTITUDE

(The figures are in terms of percentage of patients responding in the manner indicated)

SOLUTION	GRADE OF ANESTHESIA		AMOUNT OF BLEEDING DURING OPERATION			PATIENT ATTITUDE	
	NO PAIN	INJECTION REPEATED	SLIGHT	MODERATE	EXCESSIVE	CALM AND COOPERATIVE	VERY DIFFICULT
Novocain 2% with epinephrine 1:50,000	89.5 ± 1.79	5.4 ± 1.30	48.8 ± 2.99	38.3 ± 2.85	8.4 ± 1.63	40.0 ± 3.04	17.7 ± 2.36
Novocain 2% with cobefrin 1:10,000	86.7 ± 1.95	2.7 ± 0.94	35.6 ± 2.76	47.7 ± 2.88	14.4 ± 2.03	43.5 ± 2.97	16.8 ± 2.24
Probability of no difference from novocain 2% with epinephrine 1:50,000	<.001		<.001			.47	
Novocain 3% with epinephrine 1:50,000	92.9 ± 1.14	2.9 ± 0.73	28.7 ± 2.54	51.9 ± 2.79	10.8 ± 1.79	43.4 ± 2.28	11.1 ± 1.41
Probability of no difference from novocain 2% with epinephrine 1:50,000	.02		<.001			<.001	
Novocain 2%, pontocaine 0.15%, cobefrin 1:10,000	89.9 ± 1.28	4.6 ± 0.89	30.9 ± 2.67	44.6 ± 2.88	22.8 ± 2.39	48.1 ± 2.18	14.5 ± 1.53
Probability of no difference from novocain 2% with epinephrine 1:50,000	.63		<.001			<.001	

In Table III it will be noted that the percentage of patients experiencing complete anesthesia seems to be approximately the same for the four solutions. However, with the novocain 2 per cent epinephrine 1:50,000, reinjection was required in 5.4 per cent of cases. Almost identical values were obtained for the novocain-pontocaine-cobefrin mixture. With the novocain-cobefrin solution, reinjections were less numerous. With the 3 per cent novocain there was a significantly higher incidence of complete anesthesia and only a small number of reinjections. Since the differences represented only two or three patients out of each hundred, the practical significance is very low.

All three test solutions appear to produce more instances of moderate or excessive bleeding than does the reference solution in which only slight bleeding occurred in almost 50 per cent of the cases.

When the novocain strength was increased from 2 per cent to 3 per cent with epinephrine as the vasoconstrictor, the cases where there was only slight bleeding were reduced by one-half. This lends support to the suggestion made

above that the stronger novocain interferes with the vasoconstriction. Apparently the pontocaine also reduces the efficacy of the vasoconstrictor, as judged by the amount of bleeding observed. The patient attitude, such as apprehension, etc., before the injection, tended to favor the solution containing 3 per cent novocain in that a calm and cooperative patient will be less likely to manifest objective symptoms of nervousness following injection than one who is excitable. Patients who were very difficult to handle were least frequent in the strong novocain group, with the number increasing in the pontocaine group and being greatest in the novocain 2 per cent with epinephrine or cobefrin. The variability of this type of data is high and its importance in the interpretation of the general results is not entirely clear.

The observations in Tables I, II, and III of the first series were obtained by registered nurses and the operators. In order to get a still more experienced appraisal of the reactions, a second series of 622 patients was run where all the observations were made by one dental surgeon skilled in oral surgery. This dentist devoted his time exclusively to interpreting and recording manifestations of patient reactions. Therefore, all the results were comparable, as differences in individual judgment were eliminated. The data are summarized in Table IV.

The findings in Table IV are of particular interest since they serve not only to give more pointed information on patient reactions, but also to give a check reading on the volumes of solutions required to produce satisfactory anesthesia. Here again we find in a completely different set of patients that novocain 3 per cent and novocain-pontocaine-cobefrin require a lesser volume of solution to produce anesthesia than is the case with either novocain 2 per cent with epinephrine 1:50,000 or novocain 2 per cent with cobefrin 1:10,000.

In the group of patients receiving the novocain 2 per cent with epinephrine, 75 per cent had no reaction to the injection of the local anesthetic. When cobefrin was used as the vasoconstrictor in the 2 per cent novocain, 70.7 per cent had no reactions, a difference well within the limits of expected variation. It was anticipated, on theoretical grounds, that when the amount of local anesthetic in the solution was increased there might be an increase in the frequency of reactions. The difference was scarcely detectable, however, since 3 per cent novocain gave only a 71.2 per cent frequency of no reactions as compared with the 75.0 per cent control with the same vasoconstrictor.

When the cobefrin was used as the vasoconstrictor, the incidence of patients with no reactions was 70.7 per cent. Adding pontocaine 0.15 per cent to this mixture did not increase the frequency of reactions but on the contrary diminished them if anything, since now 77.0 per cent of the patients were free of reactions. The difference between these two approaches statistical significance and most clearly rules out the hypothetical possibility of an *increased* clinical toxicity from the added pontocaine.

When the individual types of reactions are considered as set forth under the headings of perspiration, tremor, respiratory embarrassment, nervousness, and tears, it is seen that increasing the novocain concentration to 3 per cent in the epinephrine series decreased the incidence of perspiration and respiratory changes but doubled the frequency of tremors and greatly increased the

TABLE IV. SUMMARY OF OBSERVATIONS IN THE 622 PATIENTS OF THE SECOND SERIES WITH REGARD TO VOLUMES INJECTED, TYPE OF INJECTION, AND FREQUENCY OF REACTION

SOLUTION	NO. OF CASES	VOLUME REQUIRED (C.C.)	TYPE OF INJECTION				PATIENT REACTION					
			INFILTRATION	INFERIOR ALVEOLAR	POST. SUP. ALVEOLAR	INFRA-ORBITAL	NO REACTION	PEESPIRATION	TREMOR	EMBARR. OF RESPIR.	NERVOUSNESS	TEARS
Novocain 2% with epinephrine 1:50,000	120	3.6 ± 0.16	52	37	11	0	75.0 ± 4.0	3.3 ± 1.6	2.5 ± 1.4	0.8 ± 0.8	15.0 ± 3.3	8.3 ± 2.5
Novocain 2% with cobefrin 1:10,000 Probability of no difference from novocain 2% with epinephrine 1:50,000	133	3.3 ± 0.12 .15	54	37	7	2	70.7 ± 3.9 .25	0.0 .03	4.5 ± 1.8 .13	0.0 .30	21.8 ± 3.6 .03	6.0 ± 2.1 .50
Novocain 3% with epinephrine 1:50,000 Probability of no difference from novocain 2% with epinephrine 1:50,000	191	2.8 ± 0.08 <.001	47	46	7	0	71.2 ± 3.3 .22	0.0 .01	5.2 ± 1.6 .02	0.0 .22	24.6 ± 3.1 <.001	6.2 ± 1.7 .50
Novocain 2%, pontocaine 0.15%, cobefrin 1:10,000 Probability of no difference from novocain 2% with epinephrine 1:50,000	178	2.8 ± 0.07 <.001	48	45	4	3	77.0 ± 3.2 .60	0.6 ± 0.6 .04	2.8 ± 1.2 .78	0.0 .23	16.9 ± 2.8 .62	3.9 ± 1.4 .13



incidence of nervousness. In contrast, producing a stronger anesthetic solution by adding pontocaine to the novocain in the presence of cobefrin did not appreciably alter the amount of perspiration and respiratory change, and actually decreased the frequency of tremor, nervousness, and tears. We do not wish to claim that these results prove novocain-pontocaine-cobefrin to be less toxic than novocain-cobefrin beyond a statistical doubt, but they make it extremely improbable that pontocaine has added to the clinical toxicity of the mixture.

#### SUMMARY AND CONCLUSIONS

Using the blind test technique, the responses of 2,382 patients to a group of four local anesthetic solutions have been studied.

The data obtained in these studies comprise observations on: volume of solution required, onset time of anesthesia, grade of anesthesia, duration of anesthesia, blood pressure changes, pulse rate changes, amount of bleeding, and patient reactions. These data were subjected to statistical evaluation. From them the following conclusions seem warranted:

1. The longest duration of anesthesia occurred with a combination of novocain 2 per cent, pontocaine 0.15 per cent, and cobefrin 1:10,000. This was significantly longer than the 3 per cent novocain with epinephrine 1:50,000 solution, and moderately longer than novocain 2 per cent with either epinephrine 1:50,000 or cobefrin 1:10,000 added.

2. The novocain-pontocaine combination and the novocain 3 per cent both required a smaller volume to produce satisfactory anesthesia than did either of the other two.

3. The differences in the degree of anesthesia produced by the four solutions, as judged by the incidence of incomplete loss of sensation, were not clinically important. All four gave generally satisfactory anesthesia.

4. The two solutions containing cobefrin appear to produce slowing of the pulse rate, whereas the two solutions containing epinephrine produce an increase.

5. The changes in both diastolic and systolic pressure are small for all of the solutions studied. No significant differences among the solutions for the circulation were found except that two of the solutions produced a slightly higher pulse pressure than did the others.

6. In general, freer bleeding occurred with the three test solutions than with the reference solution.

7. When considered from the standpoint of no reactions, perspiration, nervousness, tremor, tears, and embarrassment of respiration, it can be seen that (a) novocain 3 per cent with epinephrine differs from the reference solution in being somewhat more poorly tolerated, and (b) adding 0.15 per cent pontocaine to the novocain-cobefrin mixture does not increase, but possibly, even decreases, the incidence of reactions.

8. Each of the four solutions is a satisfactory anesthetic for the types of clinical procedures included in this investigation.

The authors desire to express their thanks to Dr. Martin E. Aronson, Dr. Eugene Schmitt, and the staffs of the Department of Oral Surgery of the College of Dentistry of New York University and of Bellevue Hospital for their valued cooperation.

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## ORAL MANIFESTATIONS OF ACUTE MYELOGENOUS LEUCEMIA

## CASE REPORT

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## HISTORY

**M**RS. A. G., aged 31 years, presented herself at the Oral Surgery Clinic of the New York University College of Dentistry for relief from persistent pain and swelling in the right maxillary region, following an extraction of an upper right second premolar three weeks previous.

*Medical History.*—There was none except for the fact that nine years ago, prior to her first pregnancy, she was told she was anemic; she did not recall any particular treatment for the condition.

*Dental History.*—Past: The patient had a full complement of teeth, except for the lower right first and second molars, and the lower left first molar which had been removed some time before. Present: About four weeks ago the patient suddenly developed swelling and pain on the upper right side of the face. She was told that she had a "gum infection" and was placed on a mouthwash. Two days later, under local anesthesia, a carious upper left second premolar was removed. The day following the extraction, the pain became increasingly severe, accompanied by temperature reading of 103° F. The patient was subsequently referred to a physician who prescribed sulfonamide therapy, 4 Gm. daily for ten days, a total of 40 Gm., along with the administration of liver and iron tablets. During this period the temperature fluctuated between 100° and 103° F. The pain did not subside completely. The tissue about the socket became puffy and the gingivae generally began to swell and hurt. Adenopathy in the submaxillary and sublingual areas be-

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came noticeable. Pain was referred to the head and neck. A general feeling of malaise and lassitude set in. Finally, in desperation, the patient went to another dentist who referred her to this institution for diagnosis.

#### EXAMINATION AND DIAGNOSIS

*Clinical Examination.*—The mucous membrane of the mouth was very pale, particularly on the palate. There was a generalized gingival hypertrophy.

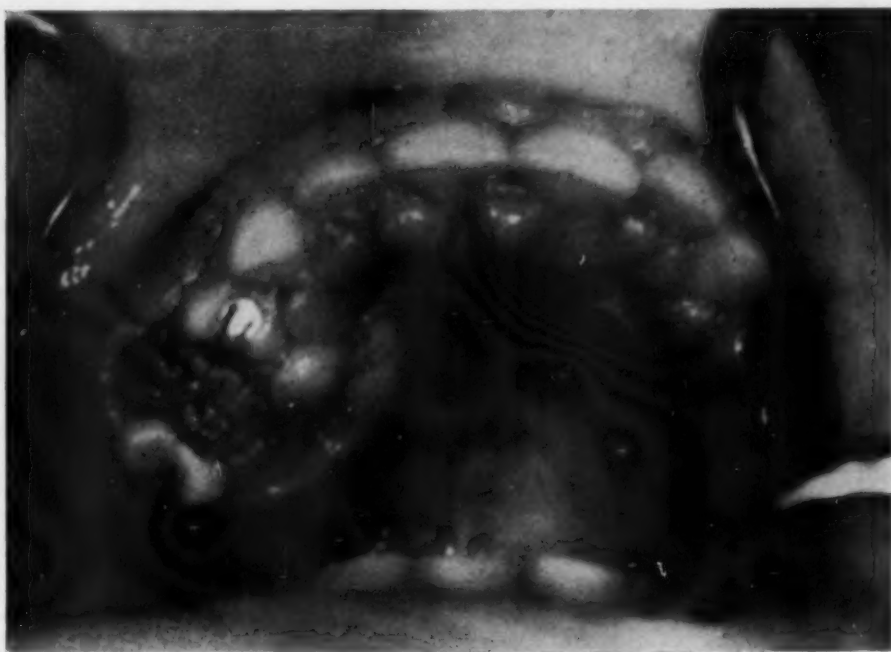


Fig. 1.



Fig. 2.

The mucous membrane, buccally and lingually to the recently extracted upper premolar socket, was markedly swollen and the socket itself was filled with exuberant granulation tissue. The right side of the face was slightly swollen. Adenopathy was not pronounced. The general appearance of the patient was toxic. An added observation was the lack of bleeding from the gums and also that the teeth were firm. (Figs. 1 and 2.)

Subjectively the patient was very uncomfortable, apprehensive, and dispirited because of her general malaise, fluctuation of temperature, and suffering from pain in both head and neck as well as in her mouth. Temperature at this time was 100.2° F.

*Clinical Diagnosis.*—A blood dyscrasia of the acute leucemic type was suspected. Laboratory tests were instituted immediately and sulfonamides were discontinued.

*Laboratory Report.*—

Wassermann reaction:

Wassermann—Cholesterolized antigen	negative
Alcoholic antigen	negative

Kahn test:	negative
------------	----------

Hemoglobin (Newcomer):	5.3 Gm.—36.7 per cent
------------------------	-----------------------

Differential blood count:

Myeloblasts ("blasts")	92
Myelocytes	2
Metamyelocytes I	1
Metamyelocytes II	1
Polymorphs	1
Lymphocytes	3
Monocytes	none seen
Eosinophiles	none seen
Basophiles	none seen

Platelets: apparently markedly decreased.

Slight qualitative changes of red cells.

*Final Diagnosis.*—Acute myelogenous leucemia. Prognosis unfavorable. The patient was referred back to her physician to whom was sent a copy of the laboratory findings.

PROGRESS

On Oct. 17, 1944, we received the following communication from the Queens General Hospital of Queens, New York:

The patient was admitted to this institution on Aug. 25, 1944, with a diagnosis of acute myelogenous leucemia. X-ray of chest: there was no evidence of any adenopathy. Lung fields were clear. Blood urea nitrogen 15 mg. per cent. Coagulation time 4 minutes. Platelet count 246,000. Blood count: hemoglobin 27 per cent; red blood cells 1,350,000; white blood cells 125,000; stab forms 14 per cent; myelocytes 28 per cent; metamyelocytes 5 per cent; premyelocytes 34 per cent; eosinophilic myelocytes 5 per cent; lymphocytes 14 per cent.

Hospital course was progressively downhill. Patient developed swelling of both parotids and a peripheral left facial palsy as well as scattered petechial hemorrhages. Gums became very hyperplastic as did tonsils and lymph glands. Kussmaul respirations were noted just before death. Expired on Sept. 17, 1944.

COMMENT

Symptoms of acute leucemia of the myelogenous, lymphatic, or monocytic type may first appear in the oral mucous membranes,<sup>1</sup> particularly in the gingivae.<sup>2-4</sup>

The sudden onset of a toothache, even in the presence of a carious tooth, accompanied by extreme pain, sharp rise in temperature, and a generalized swelling of the gums, should immediately serve as a warning.



When the socket of the suspected tooth fails to heal and the congestion, ulceration, and necrosis of the gingivae becomes marked, the presence of a blood dyscrasia is suspected.

The use of sulfonamide therapy in this instance had no effect on the rapid deterioration and ultimate result. It is possible that, by means of the sulfonamide, secondary infection of the gingivae was prevented. It is an established practice, however, to resort to frequent blood counts when sulfonamides are administered over an extended period. Had this practice been followed, the presence of the blood dyscrasia would have been detected much sooner.

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117 S. SECOND AVENUE

## SALIVARY CALCULUS IN SUBMAXILLARY REGION

## CASE REPORT

ANDREW J. BOCHICCHIO, D.D.S.,\* NEW YORK, N. Y.

A WOMAN, aged 46 years, presented at the clinic with a complaint of pain on the right side, floor of the mouth, in the region of the submaxillary gland and radiating toward the ear and mandibular region, together with vague pains along the path of innervation of the third division of the trigeminal nerve. She also experienced some pain on swallowing and occasionally felt a swelling in the posterior region, right side, floor of the mouth, which was not necessarily accentuated on eating.

A lateral radiographic examination showed what might be considered a stone either in the substance of the submaxillary gland or in Wharton's duct. An occlusal radiograph showed no evidence of a stone; however, on digital palpation its presence was definitely determined.

The question now arose as to its exact location. Was it in the duct, or was it in the gland? The answer had a bearing on the method of choice for removal. If it were in Wharton's duct it would be in a superficial position in relation to the floor of the mouth, and, therefore, the intraoral route would be the method of choice for its removal.

Further consideration led us to believe it was in Wharton's duct and it was removed intraorally as follows:

For anaesthesia, a right inferior alveolar injection was given. A 1-inch longitudinal incision was made in the mucous membrane, in the right lingual vestibule, in the floor of the mouth, midway between the third molar and the

\*Assistant Professor, Oral Surgery Department, New York University, College of Dentistry.

base of the right side of the tongue. The mucous membrane was separated and then kept open by means of Allis clamps. Then by digital palpation and blunt dissection with thumb forceps, the object was felt and removed.

Care must be taken in this surgical procedure since many important structures, such as the lingual and hypoglossal nerves, the external maxillary artery and anterior facial vein, and the stylomandibular ligament\* are in close relation. Injury to one or more of these structures, must be avoided.

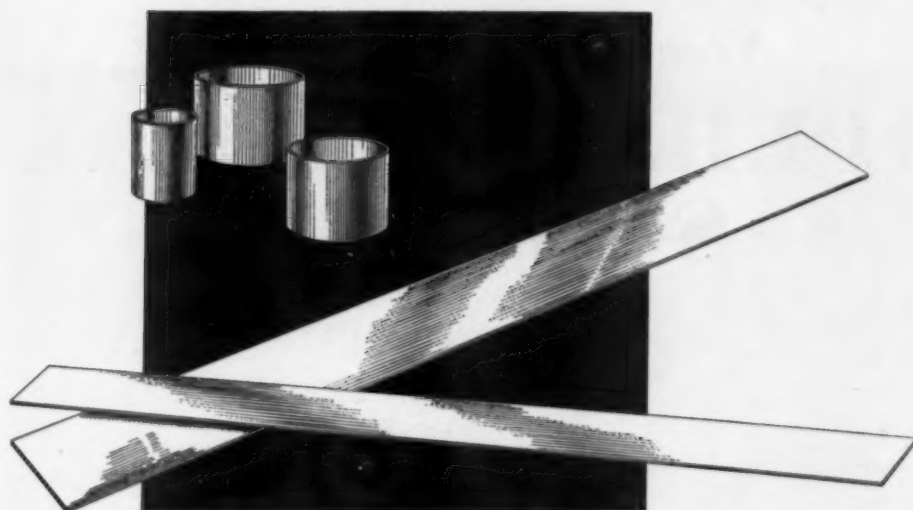


Fig. 1.

Immediately upon removal of the calculus it is necessary only to apply argyrol liberally to the wound. Postoperative care for the patient, in the nature of cold compresses externally and irrigation with warm boric acid solution internally is usually sufficient. For the relief of pain, codeine sulphate ( $\frac{1}{2}$  grain) and aspirin (5 grain) was prescribed, one every three to four hours, if necessary. Within four days, an uneventful recovery was reported.

---

\*Cunningham's Manual of Practical Anatomy, Philadelphia, 1939, Lea & Febiger, Vol. III.



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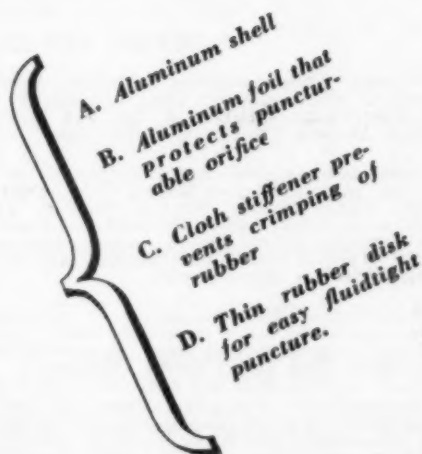
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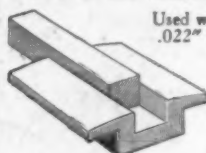
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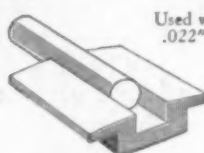
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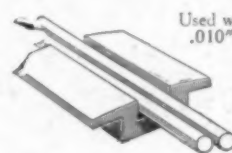
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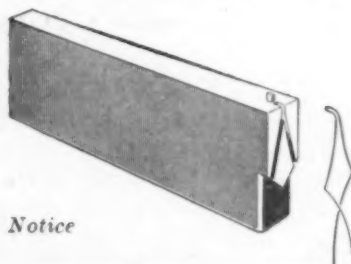
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## Original Articles

### LEUKEMIA AND DENTAL PROCEDURES

#### A REVIEW OF THE LITERATURE AND STUDY OF 123 CASES

W. F. BARNFIELD, D.D.S., M.S.\*

##### A. STUDY OF THE LITERATURE

1. *Classification and General Discussion.*—Leukemia is an invariably fatal disease of unknown etiology. The primary lesion is in the hemopoietic tissue. The disease is characterized by (1) the proliferation of cells of the leucocytic series, (2) the pervasion of the blood stream by abnormal white cells, and (3) the presence of anemia. Tumors composed of the predominating blood cells are sometimes present.<sup>1</sup> The disease is thought by some to be an infection, but at present there is more evidence that it is a neoplasm.<sup>2</sup>

Leukemia is described according to the predominating strain of cell. Subsidiary to the histological classification, and for the purposes of this paper, the reported cases and those studied are divided according to the course of the disease. In this discussion, the *form* of the disease will refer to its acuteness or chronicity, while the *type* of the disease will refer to the kind of predominating leucocyte. The differences between acute and chronic leukemia are arbitrary, and acute phases of chronic leukemia occur. The incidence of acute leukemia is greater in children, while that of the chronic form, particularly the lymphoid type, is greater in elderly persons. The onset of the acute form is slow, insidious, and, because of enlargement of the spleen and liver, often marked by a sensation of weight in the abdomen. The acute form is often fatal within a matter of weeks, while the chronic form is infrequently fatal in less than a year. The older person with chronic leukemia occasionally dies, not of leukemia, but of an infection which he has incurred because of his lowered resistance. The incipient symptoms of acute and chronic leukemia differ and depend in part upon the type of leucocyte which predominates in the blood. The symptoms and characteristics of the acute and chronic forms will be discussed separately.

*Acute Leukemia.*—While acute leukemia causes changes throughout the body, the first manifestations are frequently in the region of the head and neck. Weakness, anemia, and fever occur consistently, frequently appearing abruptly. Resistance to infection is lowered as in the chronic form of leukemia. Acute leukemia, particularly of the lymphoid and monocytic types, is often manifested by enlargement of the submaxillary, submental, and cervical lymph nodes.<sup>3, 1, 4</sup> Swelling of the lips and of the face was reported by

Prepared from a dissertation presented to the Board of Graduate Studies of Washington University, St. Louis, Missouri, in partial fulfillment of the requirements for the degree of Master of Science.

\*Instructor, the Department of Oral Pathology, University of Illinois at Chicago.

Strumpf and Daggett,<sup>5</sup> and swelling of the face has been reported by Forkner<sup>1</sup> and by Mallet and Guralnick.<sup>6</sup> Aching over the bones is an early symptom in acute leukemia. Feldman and Baskin<sup>7</sup> reported aching over the mandible in early acute monocytic leukemia. Mallet and Guralnick<sup>6</sup> reported the case of a patient with "dull aching" of the maxilla in the region of the molar teeth. Marked trismus of the mandible in a 28-year-old patient with aleukemic leukemia was reported by Negar<sup>10</sup> in 1939.

It is recognized that the first manifestations of acute leukemia often occur in the oral cavity. A study of the incidence of oral manifestations in acute leukemia based on a large number of cases was not found. Studies by Warren<sup>11</sup> and by Resch<sup>12</sup> indicate that oral manifestations are the first ones seen in about 40 per cent of the cases of acute leukemia. Warren reviewed 85 reports of acute leukemia to which he added 28 cases of his own. He found that leukemia became apparent after minor oral surgery in from 30 per cent to 40 per cent of the 113 cases. Resch<sup>12</sup> found that oral manifestations were the first symptoms of leukemia in 11 out of 21 or 52 per cent of the cases of his series. Ulceromembranous stomatitis was the manifestation most frequently found. Clough<sup>13</sup> reviewed 23 cases of monocytic leukemia and found that in 12 cases the initial symptom was gingivitis, stomatitis, or "angina." In 6 additional cases one of these symptoms appeared later during the course of the disease.

All structures in the mouth may undergo changes in acute leukemia. Four characteristics of acute leukemia bring about the lesions of the mouth and the signs and symptoms they produce. These four are: the hemorrhage into or under the mucous membrane, lowered resistance to infection, anemia, and the conspicuous infiltration of leucocytes into tissue.<sup>1</sup> The buccal mucosa may become ulcerated and hemorrhage persistently in early leukemia. Necrosis may start in the buccal mucosa,<sup>14</sup> but is likely to have been extended from the gingiva. In either case it may extend and result in a noma.<sup>15</sup>

The changes of the gingiva in acute leukemia are ulceration with necrosis, a tendency to bleed without injury, and hypertrophy with blunting of the interdental papilla. The appearance and symptoms have been described by Love,<sup>16</sup> Cook,<sup>4</sup> Forkner,<sup>21</sup> Resch,<sup>12</sup> Mallet and Guralnick,<sup>6</sup> Saghirian and Jones,<sup>17</sup> and Ambrecht and Apple.<sup>18</sup>

No description of the morphologic changes of the dental pulp in leukemia was found. Prinz and Greenbaum<sup>19</sup> stated that they saw two cases of leukemia in which "liquefaction necrosis" of the pulps took place a few months before death. Reports of symptoms referable to the teeth indicate that the dental pulps occasionally undergo leukemic changes. A patient reported by Feldman and Baskin<sup>7</sup> experienced pain which he attributed to "several bad teeth" which he wanted extracted. It soon became evident that the patient had acute leukemia. Kuhn, Helwig, and Webb<sup>20</sup> reported that a boy with lymphoid leukemia had a toothache. No apparent reason for pain was given. Forkner<sup>21</sup> stated that patients with acute leukemia sometimes have aching teeth as an early manifestation of the disease. Contrary to this observation was the comment by Reznikoff<sup>22</sup> that "it is not my impression that leukemic patients complain of aching teeth more frequently than of other pain." Dr. C. V. Moore<sup>23</sup> was of the same opinion. Following this writing I have received a personal



communication from Dr. Lester Burket stating that consistent changes in the pulp have been observed and will soon be described in the literature by him.<sup>23a</sup>

The changes which gingival and periodontal tissue undergo in leukemia are essentially the same as those found in other tissues. Goldman<sup>24</sup> described the periodontal tissues in leukemia as being fibrotic and necrotic in places, and studded with myelocytes. Gingival tissue in leukemia, as described by Prinz and Greenbaum,<sup>19</sup> is heavily infiltrated with leukocytes.

The changes of the periodontal tissues may produce symptoms which appear to be of dental origin. Edsall<sup>25</sup> in 1905 pointed out that it had been suggested that the rapidity of the progress of leukemia was frequently caused by oral infection and reported a case of acute leukemia in which swelling of "the gum about the posterior molar tooth" apparently was the reason for extraction. Hopper<sup>26</sup> reported the case of a boy who had pain around a lower premolar which was treated by a mouthwash until a diagnosis of leukemia was made. In the case reported by Feldman and Baskin,<sup>7</sup> pain and swelling were present over the mandible. It is not possible to decide after reading the report whether the lesion producing the pain was in the mandible or in the teeth. Osteomyelitis of the maxilla in leukemia was reported by Grawitz,<sup>27</sup> Thieleman,<sup>28</sup> and Ulses.<sup>29</sup> Forkner<sup>1</sup> stated that osteomyelitis sometimes followed extraction.

*Chronic Leukemia.*—A consideration of chronic leukemia is also of importance to this study. The symptoms which mark the gradual onset of the chronic form of leukemia are due to four characteristic changes: hypermetabolism of the patient, enlargement of the spleen, increased tendency to bleed, and anemia. Increase in metabolic rate accounts for sweating, nervousness, and fatigability. The enlargement of the spleen sometimes produces a sensation of weight in the abdomen. The tendency to bleed without cause accounts for bleeding from the gums, and the anemia is responsible for the weakness of the patient.

In many ways chronic leukemia does not differ greatly from the acute form. For example, the incipient symptoms of the two forms may be similar. Enlargement of the lymph nodes in chronic leukemia, particularly in the lymphoid type, is commonly seen late in the course of the disease, and may be seen early.<sup>1</sup> The oral manifestations of acute leukemia have been pointed out repeatedly in dental literature. Chronic leukemia likewise has manifestations which concern the dentist. "Trench mouth" with hypertrophy of the gingiva and enlargement of the submaxillary lymph nodes following extraction was pointed out by Resch in his third case.<sup>12</sup> A "gumboil" which contained only blood (lymphoma) in a patient with chronic leukemia was described by Resch in his fourth case.

Love,<sup>16</sup> in the section of his paper dealing with lesions of the mouth, described a man with chronic myeloid leukemia who probably had teeth extracted hoping to check his loss of weight, loss of appetite, and weakness. Love pointed out in his discussion that extraction was often undertaken, hoping for improvement from recent ill-health.

In summary, two points have been brought out by a review of the literature concerning acute and chronic leukemia. First, oral manifestations of acute leukemia may resemble those of diseases of the mouth. Toothache,

ulceromembranous stomatitis, enlargement of the submental, submaxillary, and cervical lymph nodes, swelling of the lips and face, and pain in the mandible are usually of local origin. It has been shown that each of these symptoms may be a manifestation of leukemia. Second, as Love inferred, the symptoms of chronic leukemia may be confused with those attributed to disease caused by foci of dental infection. Infected teeth are removed, and rightfully, in the treatment of chronic diseases associated with the streptococcus, such as proliferative arthritis. Likewise the extraction of teeth, either periodontally or periapically infected, is sometimes suggested as a general measure to improve the health of patients suffering from anemia, loss of weight, and loss of strength. The possibility that these symptoms might be signs of chronic leukemia is overlooked.

2. *Literature Concerning the Relation of Dental Procedures to Leukemia.*—

An extended discussion of the relation between dental procedures and leukemia was not found. Some evidence was gathered from the brief discussions by writers reporting cases of leukemia, but the bulk of information was obtained by an analysis of the case reports found in the literature in which dental procedures were associated with leukemia. (When a dental procedure was performed immediately before or during manifestations of leukemia, it was said to be associated with the leukemia.)

A statement as to the role of extraction in leukemia usually concerned only the case reported. Many instances were reported in which extraction was associated with the leukemia and the relation of the extraction to the leukemia was not considered. Other cases of leukemia associated with extraction were reported because the systemic disease first became manifest after the extraction of teeth.

Three differing ideas were found in the published discussions concerning the relation between dental procedures and leukemia: (1) dental procedures were performed because of symptoms of leukemia, (2) extraction of teeth was in some way operative in changing chronic to acute leukemia, or in "precipitating" an acute phase of the disease, and, (3) the oral infection which was observed sometimes following an extraction was a possible cause of acute leukemia. A discussion of these three clinical impressions of the role of extraction in leukemia follows.

The earliest report found in which the writers felt that extraction was performed because of a manifestation of leukemia was published in 1902 by Hirschfeld and Alexander.<sup>30</sup> A patient was described in whom the incipient symptom was "swelling in the foot." Soon after, a swelling of the under jaw came on, "*treated*\* by extraction of the tooth." Eggena<sup>31</sup> made no specific statement, but commented that patients often consulted the dentist for treatment of a condition that seemed to be a local disease, but was, as shown by its course, actually leukemia. Love<sup>16</sup> remarked that teeth often were extracted with the idea that it might improve poor health, but was not more specific as to the role of extraction. Forkner<sup>21</sup> stated that patients with leukemia often consulted a dentist because of bleeding gums or aching teeth thought to be of local origin. He believed that extraction or other dental procedures were sometimes performed in the belief that the proper therapeutic measures were being taken.

\*Italics mine.

The second opinion, that chronic leukemia was "transformed into an acute affair" by the extraction of teeth, was mentioned by Edsall<sup>25</sup> in 1905 and was expressed by Isaacs<sup>32, 33</sup> in 1925 and 1926. He stated that "he had in mind" several such instances and reported one (Report 18 in Table II). No explanation of the manner in which the leukemia might be changed in form, or evidence of the occurrence was presented.

Reference will be made later to the suggestion of Hill<sup>34</sup> that the infection which followed the extraction of teeth was a cause of leukemia.

Further inquiry was made into the relation of dental procedures to leukemia by an analysis of the reports presented in the literature in which dental operations were associated with leukemia. (Later in the study the denial of the causal relation of extraction to leukemia was questioned, discussed and, it was felt, verified.) The premise that leukemia does not result from extraction made necessary a second postulate: that leukemia, which became apparent immediately after extraction, was present before the procedure.

In order to study the relation between extraction and leukemia, all of the reports that could be found in which extraction was in association with leukemia were summarized in tabular form. A total of 33 such case reports was found. Aside from the data necessary to study the relation of the procedure to leukemia, comments and conclusions concerning the relation were included.

Evidence from the literature seemed to suggest a bilateral relation between leukemia and dental procedures. On the one hand, Isaacs stated that dental procedures, specifically extraction, sometimes had a bearing on the course of the disease. On the other hand, Forkner along with Osgood<sup>35</sup> found that patients with acute leukemia went to the dentist complaining of dental disease, for which procedures were performed. After learning something of the general nature of leukemia and reviewing reports, it occurred to me that older persons might consult a dentist because of vague symptoms marking the onset of chronic leukemia. It is a common observation that many older people are conscious of having teeth of questionable benefit to their health, and that vague symptoms of systemic disease elicit a procrastinated visit to the dentist. It is natural that the teeth should, under these conditions, be regarded as a contributory factor to the systemic complaints.

If the relation suggested by Forkner existed, a study of the symptoms causing the patient with leukemia to visit the dentist should produce pertinent information. Following up this idea, the complaints in each case report were noted. An approach to the relation suggested by Isaacs, that extraction sometimes changes the course of the disease, was made by noting the symptoms and severity of the disease before and after the oral operation.

Of the two relations being investigated, the more subtle was Forkner's suggestion that complaints of leukemia which seemed to be of dental origin were treated by dental manipulation. To facilitate the study of this relation the tabulated reports were separated into three groups on the basis of the complaints for which dental treatment was sought. Seventeen reports in which the reason for the procedure actually was stated or was evident by deduction were placed in Table I. Six additional reports in which the reason for the operation was fairly clearly indicated or could be deducted with reasonable

TABLE I. REPORTS IN WHICH THE REASON

NO.	YEAR	AUTHOR AND CASE	AGE, SEX, FORM AND TYPE OF LEUKEMIA	MANIFESTATIONS OF LEUKEMIA PRESENT BEFORE EXTRACTION
1	1902	Hirschfeld, Alexander <sup>30</sup>	21 M Acute Monocytic	"Swelling in foot" preceded swelling of jaw
2	1905	Ewald <sup>36</sup>	60 M Acute Myeloid	Pain in tooth roots of upper left jaw. Abscess formed which drained spontaneously
3	1926	Hill <sup>34</sup> Case 1	68 M Acute Myeloid	"Toxic spells"
4	1929	Eggens <sup>31</sup> Case 1	34 M Acute Lymphoid	Eggens indicates in discussion that patient had leukemia before extraction
5	1932	Sydenstricker, Phinzy <sup>37</sup>	47 F Acute Monocytic	"Bluish spots over thighs and legs." Felt weak, ill, dyspneic on exertion
6	1933	Kuhn, Helwig, Webb <sup>20</sup> Case 5	24 M Acute (?) Monocytic	Gingivitis, loss of weight and strength, "run down." 14,000 leucocytes, 37% myelocytes (monocytes)
7	1934	Forkner <sup>38</sup> Case 3	40 M Acute Monocytic	"Perfectly well"; gingiva bled and became swollen and tender
8	1934	Doan, Wiseman <sup>39</sup> Case 1	66 M Chronic Monocytic	No statement as to health before first extraction. Total leucocytes 12,480. Monocytes 36% at time of second extraction
9	1934	Doan, Wiseman <sup>39</sup> Case 1	63 M Chronic Monocytic with aleukemic phase	Weak, anemic, leukemia cutis. Monocytes 30% of 5,000 leucocytes
10	1934	Doan, Wiseman <sup>39</sup> Case 6	34 F Acute Monocytic	Tonsillitis followed by otitis media. Gingivitis
11	1935	Mann <sup>40</sup> Case 1	57 M Acute Monocytic	Sore throat, oral lesion, general malaise
12	1936	Klumpp, Evans <sup>41</sup> Case 3	12 M Acute Monocytic	Loss of appetite and weight. Drowsiness, pallor, constant pain in right upper quadrant



## FOR THE DENTAL PROCEDURE IS CLEAR

DENTAL PROCEDURE; REASON FOR PROCEDURE	INTERVAL BETWEEN EXTRACTION AND APPEARANCE OF THESE SYMPTOMS. SYMPTOMS OF LEUKEMIA WHICH CLOSELY FOLLOWED EXTRACTION	COMMENTS AND CONCLUSIONS
Extraction to "treat" swelling of jaw	Profuse bleeding and "ulcer" formed over extraction wound. Submaxillary lymph nodes became enlarged	Word "treat" taken from report. Swelling of jaw, like that of foot, was manifestation of leukemia
Extraction of roots to eliminate periapical abscess	Immediately, profuse bleeding and anemia; gingivitis	Leukemia was present at time of extraction; may have been responsible for exacerbation of chronic periapical abscesses
Extraction to relieve "toxic spell"	3 days. Chilly sensations, prostration, fever, swelling and tenderness of submaxillary lymph nodes. Area around alveolus was swollen, bluish, and did not heal	Dentist finally persuaded patient to have teeth extracted as this might relieve "toxic feeling." As manifestations of leukemia followed extraction closely, it is concluded that toxic feeling was symptom of leukemia
Extraction. It is indicated in discussion that extraction was performed because of an oral manifestation of leukemia	Immediately. Excessive bleeding	Extraction was performed because of signs thought by dentist to be local in origin, but actually those of leukemia. This conclusion is based on Eggena's discussion
Extraction of right upper premolar tooth because of acute toothache. Tooth found to be abscessed	5 days later. Developed a marked painless swelling. Hearing impaired. Weakness progressed	Not possible to say if abscess of tooth was associated with leukemia or not
Extraction to treat gingivitis	"Followed." Gingivitis progressed to become necrotizing stomatitis which ultimately resulted in necrosis of maxilla	Extraction was performed to treat progressing symptom of leukemia
Extraction to relieve pain in region of second left upper molar	Excessive hemorrhage resulting in serious loss of blood. Ulceration of gingiva	Leukemia was first manifested by swollen, tender gingiva. Tooth was extracted because of aching caused by leukemia
Extraction of 12 upper teeth on Jan. 28 to remove foci of infection. Extraction to eliminate "obvious foci of infection"	Hemorrhage, to point of exsanguination. Weakness for months. One week following second extraction, severe hemorrhage for 24 hours. Total count 26,900. Monocytes 65% after second extraction	Patient probably had first extraction because he was not feeling well and was hoping that removal of teeth would improve general health
Extraction of molar because of "foci of infection"	No untoward sequelae	Extraction performed after knowledge of leukemia, and probably without relation to leukemia
Incision of 7 oral abscesses within 2 weeks	Necrotizing osteomyelitis of maxilla. Acutely ill. Of 99,000 leucocytes, 68% were immature monocytes	Patient had unrecognized leukemia at time of dental treatment
Extraction of upper right third molar because of "small necrotic patch seen around root"	Gangrene, right side of palate. Tumors of leukemic cells appeared under skin. Death in 3 days	General malaise was sign of leukemia. Either sore throat or dental extraction made leukemia much more severe
Extraction because of pain interpreted as toothache, present on both sides of lower jaw	Gingiva became swollen, red, tender, progressed to ulceration and necrosis	Pain on both sides of lower jaw was interpreted as toothache. Pain was in all probability caused by leukemia

TABLE I.

NO.	YEAR	AUTHOR AND CASE	AGE, SEX, FORM AND TYPE OF LEUKEMIA	MANIFESTATIONS OF LEUKEMIA PRESENT BEFORE EXTRACTION
13	1936	Klumpp, Evans <sup>41</sup> Case 6	16 M Chronic Monocytic	High fever, sweats, loss of weight. Patient in bed
14	1936	Love <sup>16</sup> Case 12	27 M Acute Myeloid	Ulcer in mucous membrane of the mouth, posterior to tooth. Gums were sore
15	1936	Love <sup>16</sup> Case 14	50 M Chronic Lymphoid	Swollen lymph nodes of neck, bilateral, for several months. Recent swelling of right side of lower jaw
16	1940	Resch <sup>12</sup> Case 4	40 M Chronic Monocytic	No systemic evidence of leukemia stated. "Gumboil" of mouth was probably lymphoma
17	1941	Friedenberg <sup>42</sup>	(Not stated) Chronic Myeloid	Diagnosis by clinical and hematological studies

TABLE II. REPORTS IN WHICH THE REASON FOR THE

NO.	YEAR	AUTHOR AND CASE	AGE, SEX, FORM AND TYPE OF LEUKEMIA	MANIFESTATIONS OF LEUKEMIA PRESENT BEFORE EXTRACTION
18	1926	Isaacs <sup>32, 33</sup>	61 M Chronic (said to assume acute form) Lymphatic	Weakness
19	1933	Kuhn, Helwig, Webb <sup>20</sup> Case 6	51 M Acute Lymphoid	Soreness of mouth which progressed. Mouth became swollen after extraction
20	1934	Forkner <sup>38</sup> Case 4	26 M Acute Monocytic	Report was confusing; stated that patient "felt quite well until toothache." Later, "had not felt as strong as previously—suffered from recurrent attacks of abdominal pain"
21	1935	Whitby <sup>44</sup> Case 1	44 F Chronic Monocytic	Was diagnosed on basis of hematological and clinical studies
22	1935	Love <sup>16</sup> (See under section on "the mouth" in Love's paper) Case 8	43 F Chronic Myeloid	Mass in abdomen. Loss of weight, appetite, and strength. Mass was reduced in size after post-extraction hemorrhage
23	1940	Resch <sup>12</sup> Case 2	45 M Chronic Monocytic	Report stated that patient was in good health at time teeth were extracted

DENTAL PROCEDURE; REASON FOR PROCEDURE	INTERVAL BETWEEN EXTRACTION AND APPEARANCE OF THESE SYMPTOMS. SYMPTOMS OF LEUKEMIA WHICH CLOSELY FOLLOWED EXTRACTION	COMMENTS AND CONCLUSIONS
Extraction of one tooth. Tooth-ache	Fetor oris (no more information given)	No indication as to cause of "toothache"
Extraction to relieve pain and as measure to reduce infection	Alveolus bled excessively. Gingivitis became rapidly severe	On basis of experience, conclusion is drawn that ulcer posterior to third molar tooth was believed to be operculum, which would heal if tooth were removed
Extraction to treat swelling of jaw	Swelling was not improved. Area around alveolus became ulcerated	
Incision. Tender swelling in maxillary buccolingual fold was diagnosed as "gumboil" and opened	Continuous bleeding for 15 days. Necrosis of buccal mucosa	"Gumboil" contained "only blood," and was lymphoma
Removal of 6 upper anterior teeth	Healing was normal and without untoward results	

## DENTAL PROCEDURE IS INFERRED, OR MAY BE DEDUCTED

DENTAL PROCEDURE; REASON FOR PROCEDURE	INTERVAL BETWEEN EXTRACTION AND APPEARANCE OF THESE SYMPTOMS. SYMPTOMS OF LEUKEMIA WHICH CLOSELY FOLLOWED EXTRACTION	COMMENTS AND CONCLUSIONS
Extraction of 7 teeth because of pyorrhea; removed to eliminate foci of infection	Few days. Coughed up blood. Throat became sore. Patient became weaker	In view of patient's age and weakness, and extraction of 7 teeth within a short time, it is reasonable to assume that teeth were pyorrhetic and were extracted hoping to improve the general state of health
Extraction—to treat pyorrhea	Next day. Chills and sweating every day for a week. Mouth became extremely sore and swollen	
Extraction—to relieve toothache	Profuse hemorrhage, patient had grown progressively weaker	Patient had leukemia at time of toothache. It is possible, although not evident, that teeth were taken out because of signs of leukemia
Extraction of upper molar tooth. Apparently teeth were suspected as being foci of infection	Patient was improved at time of extraction. "Within a few days" ulceration around tooth reappeared. Patient became ill, and had second attack	Extraction of tooth seemed to cause leukemia to become severe and more progressive
Extraction of 3 teeth to relieve weakness, "run down" feeling	Bled profusely	Reason for extraction was not stated. In general discussion, Love stated that extraction was "undertaken with the idea that it might improve the patient's health"
Extraction of 5 teeth because of pyorrhea	Bleeding from alveoli was intermittent from time of extraction	Patient was not followed up, and diagnosis not confirmed as one of leukemia. Anemia was marked. Total enumeration of leucocytes was normal, but differential showed 55% were monocytes

TABLE III. REPORTS IN WHICH IT IS IMPOSSIBLE TO GAIN BY INFERENCE

NO.	YEAR	AUTHOR AND CASE	AGE, SEX, FORM AND TYPE OF LEUKEMIA	MANIFESTATIONS OF LEUKEMIA PRESENT BEFORE EXTRACTION
24	1902	Grawitz <sup>27</sup>	42 F Acute Mixed (?)	Health was previously good
25	1926	Hill <sup>34</sup> Case 2	35 F Acute Myeloid	Patient was strong before extraction. Indication that there was gingivitis present at time of extraction
26	1929	Walmesley <sup>47</sup>	17 F Acute Lymphatic	No indication
27	1930	Dameshek <sup>43</sup>	50 M Monocytic	"Felt well"
28	1933	Sergeant, Laugier <sup>45</sup>	M Acute Myeloid	No indication of health
29	1934	Forkner <sup>38</sup> Case 7	17 M Acute Myeloid	Good dental history not obtainable. Appeared to be well at time tooth was extracted
30	1936	Abel <sup>46</sup>	17 M Acute Lymphoid	Tonsils were removed without incident a "short time before" extraction
31	1936	Klumpp, Evans <sup>41</sup> Case 8	74 F Acute Monocytic	No indication as to state of health before extraction
32	1937	Forkner <sup>48</sup>	33 M Acute Monocytic	Was "well" before extraction of tooth
33	1940	Resch <sup>12</sup> Case 3	41 F Chronic Monocytic	No indication

certainty were put in Table II. Ten reports in which the reason for the procedure could not be determined because of lack of detail were placed in Table III.

Examples of reports placed in the first two groups with the reasons for so placing them are submitted. An example of a report in which the reason for extraction was clear although not stated is Report 15 (Love's Case 14). The patient was a 50-year-old man. A swelling of the right angle of the mandible was painful at times. The patient had two lower right teeth extracted, "which did not ease the pain." It is a common observation that the cervical lymph nodes located just under the angle of the mandible become swollen and painful as a result of periapical infection of the lower molar teeth. The gen-



OR BY REASONING, THE PURPOSE FOR THE DENTAL PROCEDURE

DENTAL PROCEDURE; REASON FOR PROCEDURE	INTERVAL BETWEEN EXTRACTION AND APPEARANCE OF THESE SYMPTOMS. SYMPTOMS OF LEUKEMIA WHICH CLOSELY FOLLOWED EXTRACTION	COMMENTS AND CONCLUSIONS
Extraction of 14 teeth	Long-continued hemorrhage. Necrosis of jaw, enlargement of spleen and liver	Extraction of 14 teeth at one sitting suggests that teeth were removed because of pyorrhea
Extraction of 1 tooth	Pain in jaw and ear on side of extracted tooth. Ulceromembranous stomatitis continued to progress	
Extraction	Immediately following extraction there was long and continued bleeding	
1926. Extraction abscesses Oct., 1927. Submaxillary gland removed because of stone Sept., 1928. Extraction of 4 lower teeth	"Didn't feel well" Felt depressed. "Pain in flank"  Gingivitis, alveoli did heal. Oct., 1928, leukemia diagnosed	Difficult to determine onset of leukemia
Extraction	Immediately, persistent hemorrhage of alveolus, followed by anemia, swelling of the face	
Extraction	Profuse and prolonged bleeding from alveolus. Weakness	No clue to reason for extraction
Extraction	Excessive hemorrhage followed extraction. This continued until death	
Extraction	Gums became swollen, ecchymotic, and inflammation extended. Inflammation slowly completely healed	As no clue to cause of extraction was stated, impossible to determine what relation extraction had to leukemia except that it made leukemia manifest
Extraction of upper molar	Small amount of bleeding from alveolus for 2 days. Sore throat followed 2 or 3 days after extraction. Pharyngitis subsided, but was followed "soon afterwards" by anorexia, weakness, and listlessness	Not enough evidence to conclude that leukemia was present at time of extraction. Amount of bleeding and sore throat indicated are not unexpected following removal of upper third molar
Extraction	"Trench mouth" 2 days after extraction. Hypertrophy of gingiva and submaxillary lymph nodes	

eral treatment for dental infection is removal of the tooth. The statement that the extraction "did not ease the pain" strongly implies that relief of the pain was expected from extraction. It was concluded that the purpose of the extraction was clear and this report therefore was placed in Table I.

An example of a report placed in Table II because the purpose of extraction was indicated fairly well, but not stated, is Report 22 (Love's Case 8). The patient was a 43-year-old woman. The complaints were weakness, loss of weight, and anemia. A physician was treating the patient when she "was advised to have some teeth extracted." Following the removal of three teeth, the obvious signs of leukemia became manifest. I drew the conclusion that the physician,

hoping that extraction of infected teeth would improve the patient's general health, had ordered the procedure.

As the reason for placing the reports in the third group was lack of detail, it seems unnecessary to proffer an example of a report from Table III.

3. *Analysis of the Collected Reports.*—After the 33 collected reports were tabulated and separated into groups, they were examined as to (1) the presence or absence of leukemia before the procedure, (2) the effect of the extraction on the course of the disease, and (3) the complaints for which the procedures were performed. Of the 28 reports in which the state of health before extraction was indicated, there was some indication that leukemia was present before extraction in 23, or 82 per cent. These reports were Numbers 1 to 23, inclusive. (In Report 20 leukemia was evidently present before the extraction in September, 1928, although probably not present at the time of the previous dental procedure.) In 5, or 18 per cent, of the reports there was no evidence that leukemia existed before extraction. These reports were Numbers 24, 25, 27, 30, 32. In the five reports in which the state of health before extraction was not indicated, there was not enough evidence to judge whether or not leukemia was present at the time of extraction. These reports were Numbers 26, 28, 29, 31, and 33.

The relation of extraction to leukemia suggested by Isaacs, that extraction sometimes "precipitated" chronic leukemia, was analyzed by studying the indications as to the severity of the disease before and after extraction. It was assumed that leukemia which became manifest immediately following extraction was present at the time of extraction. Isaac's assertion must be interpreted before evidence can be weighed. As has been indicated earlier, the acuteness or chronicity of leukemia is arbitrary. The expected length of life of the patient is foremost in the clinician's mind in describing a case as acute or chronic.

The criterion established in order to determine whether or not a report supported Isaacs' opinion was as follows: when any changes following extraction appeared to shorten the disease, the report was considered as supporting Isaacs' assertion.

There was sufficient evidence to judge the effects of extraction on the course of the leukemia in 15 of the 33 reports. In 18, the evidence was not sufficient to permit any judgment. Of the 15 reports, in 10, Numbers 6, 7, 8, 10, 11, 12, 21, 24, 28, and 29, the leukemia seemed to become more acute following the dental procedure. In 3 reports, Numbers 1, 2, and 3, the procedure probably made the course of the disease more acute, although the evidence was not as strong as in the above 10 reports. In 2 reports, Numbers 9 and 17, the extraction seemed to have no effect on the leukemia.

The tabulated reports in Tables I and II were examined in the light of Forkner's suggestion, that dental procedures are performed on patients with leukemia because of the oral manifestations of the disease. In 16 of these 23 reports, a decision was made as to whether the complaints for which the extraction was performed were manifestations of leukemia or not. In 14, or 88 per cent of these 16, manifestations of leukemia seemed to cause the complaints for which the procedure was performed. The numbers of these 14 reports divided as to type or form are set forth in Table IV.

TABLE IV. REPORTED CASES IN WHICH THE COMPLAINTS FOR WHICH EXTRACTION WAS PERFORMED WERE MANIFESTATIONS OF LEUKEMIA; ARRANGED AS TO TYPE AND FORM

<i>Lymphoid Leukemia</i>
Acute—Report 4
Chronic—Report 15
<i>Myeloid Leukemia</i>
Acute—Reports 3 and 14
Chronic—Report 22
<i>Monocytic Leukemia</i>
Reports 1, 6, 7, 8, 10, 11, 12, and 16
Undetermined as to form—Report 18

As mentioned earlier, in Reports 9 and 17 the extraction was independent of the systemic disease.

In the 7 reports remaining of the total 23, it was impossible to judge the cause of the complaints for which extraction was performed. From evidence obtained from brief discussions in the literature, and from an examination of the reports, it appeared that the manifestations of leukemia were sometimes the reason for performing dental procedures. Again, procedures changed the course of the systemic disease. More evidence as to this relation was gained from the studied cases than from the collected reports because of the greater detail available in the clinical records.

#### B. MATERIALS AND METHODS

Cases of leukemia for study were found by searching the protocols of 10,300 autopsies performed by the Department of Pathology of Washington University. The hospital records of the cases of leukemia were read for the purpose of finding: (1) the type and form of each case of leukemia, and (2) whether or not a dental procedure was associated with the systemic disease. In a few cases the type was not stated and no further effort was made to establish it. The case was then classified as "type undetermined." Proceeding on the thesis that aleukemic leukemia and leucosarcoma did not differ essentially from leukemia, special note of these types was not made. For the purpose of this study, the cases were classified as to their form. Cases described in the clinical record as "subacute" were grouped with the cases of acute leukemia. When no statement was found concerning the form of the disease, the following characteristics were considered in making a decision. It was decided that the leukemia was acute if: (1) The onset was abrupt. (2) The disease lasted less than six months. (3) The predominating leucocyte was immature in form. (4) The hemoglobin and platelet count fell rapidly at the onset of the disease.

A case was selected for this study when a dental procedure was performed on the patient, or when manifestations appeared immediately after the procedure.

#### C. ANALYSIS OF THE DATA

A tabulation of the cases of leukemia analyzed first as to their bearing on the problem at hand, and second as to their type and form, is given in Table V.

Of the total 123 cases of leukemia, 62 were acute, of which 7, or 11.2 per cent, were selected for this study, and 57 were cases of chronic leukemia, of which 3, or 5.3 per cent, were selected. A numerical comparison of the cases studied to those not included for study, as to the type of leukemia, was made. The total number of cases of monocytic leukemia was 8 and of these, 2, or 25 per cent, were selected for this study. Of the 123 reports, 58 were lymphoid,

TABLE V. CASES OF LEUKEMIA FROM AUTOPSY PROTOCOLS

I. CASES NOT SELECTED FOR THIS STUDY		
A. Lymphoid leukemia		54
Acute	32	
Chronic	22	
B. Myeloid leukemia		49
Acute	19	
Chronic	30	
C. Monocytic leukemia		6
Acute	4	
Chronic	2	
D. Undetermined		4
Total		113
II. CASES SELECTED FOR THIS STUDY		
A. Lymphoid leukemia		4
Acute (CC; DD; EE; KK)	4	
B. Myeloid leukemia		4
Acute (FF)	1	
Chronic (AA; BB; GG)	3	
C. Monocytic leukemia		2
Acute (HH; JJ)	2	
Total		10
Grand Total		123

of which 4, or 6.9 per cent, were selected for study. Myeloid leukemia appeared 53 times and of these cases 4, or 7.5 per cent, were pertinent.

The evidence thus far gained from the review of the literature had pointed to a double relation between extraction and leukemia. It appeared that because of leukemia, dental procedures were sometimes performed, and also that extraction had a bearing on the course of the disease. In order to determine whether data from the studied cases might elucidate these relations, Table VI was set up.

The same general information was tabulated in Table VI as in Tables I, II, and III. Aside from basic information relative to the patient, local or systemic signs of leukemia present before extraction were noted. An effort was made in studying each of the cases to find the motive for the dental procedure, and this was recorded in the table. In order that the postextraction changes due to the leukemia might be studied, information relative to these was also entered as "Reaction of the oral tissue to dental procedure." The table was concluded with comments concerning the relation of leukemia to the procedure.

#### D. DISCUSSION

1. *Frequency of Extraction.*—There is no precise information as to how often dental extraction must be or has been done in patients with leukemia. Warren indicates that from 30 to 40 per cent of all patients with acute leukemia have prolonged hemorrhage following minor surgery including tonsillectomy and dental extraction. In the present study, dental manipulation was associated with leukemia in nearly 1 case in 12, or 8.3 per cent. In comparing these figures with those of Warren, it must be remembered that in the present study dental procedures were the only form of minor surgery considered. Since Warren had included other forms of surgery, it is not surprising that his percentages are somewhat higher.



As was indicated in the review of the literature, the acute form of leukemia is associated with extraction more often than is the chronic form. The number of cases of acute leukemia in the collected reports was 21, of chronic leukemia, 10. In 3 of the collected reports, there was doubt as to the form of the leukemia.

It was emphasized by Forkner and by Clough that monocytic leukemia was manifested by oral changes more frequently than any other type. If oral signs were more frequent, a higher percentage of cases of monocytic leukemia would be expected in association with dental procedures. Table VII presents the percentage of selected studied cases of each type compared to the total number of cases of that type of leukemia.

The strikingly higher percentage of studied cases of monocytic leukemia associated with extraction is in agreement with the analysis of the collected reports. Of the 14 collected reports in which leukemia seemed to cause the complaint for which extraction was performed, 7, or half, were of monocytic leukemia.

2. *Effect of Dental Procedures on Leukemia.*—The most frequently observed relation of leukemia to extraction is the conspicuous manifestation of leukemia following dental operations. In 8 of the 10 pertinent cases, oral changes were described which followed extraction and were due to leukemia. Necrosis, hemorrhage, swelling of the gingiva, or other obvious manifestations followed the extraction in 7 of these 8 cases, Cases AA, CC, DD, EE, FF, HH, and JJ. In 31 (all except Numbers 9, 18, and possibly 32) of the collected reports, conspicuous local, and, in some reports, general changes followed the manipulation.

As has been stated, it was assumed as a working hypothesis that dental manipulation did not cause leukemia. This premise must be examined and found to be sound if the study based on it is to stand. The prompt appearance of secondary infection following extraction in patients with leukemia suggested to Hill that oral infection was the cause of leukemia. The indication that there were present before extraction, signs or symptoms, either oral or general, of leukemia constituted evidence that leukemia was present before extraction.

In 7 of the studied cases (AA, CC, DD, EE, FF, HH, JJ) frank signs of leukemia followed dental manipulation. In 5 of these 7, evidence was found that leukemia probably was present before the dental manipulation. These 5 cases, and the symptoms of leukemia existing before extraction follow:

AA—Weakness.

DD—Increasing weakness.

EE—Irritability, swollen submaxillary lymph nodes, gingivitis.

FF—Gingivitis.

HH—Felt "below par," had not recovered from "flu" in a reasonable length of time.

It is presumed that in cases CC and JJ leukemia was present, but not manifest until after extraction.

When the collected reports were considered for evidence of the presence or absence of leukemia before extraction, it was found that in 23, or 82 per cent, of the 28 reports in which the state of health previous to extraction was

TABLE VI. FACTS RELATIVE TO THE RELATION BETWEEN LEUKEMIA AND DENTAL PROCEDURES IN THE STUDIED CASES

CASE, TYPE, AND FORM	EVIDENCE AS TO GENERAL HEALTH OF PATIENT BEFORE EXTRACTION	CONDITION OF TEETH AND MOUTH BEFORE EXTRACTION	PROCEDURE AND REASON FOR PROCEDURE	REACTION OF ORAL TISSUE TO DENTAL PROCEDURE	PROBABLE RELATION BETWEEN PROCEDURE AND LEUKEMIA
AA Myeloid Chronic	Weakness present	Loosening of teeth. Osteomyelitis, necrosis of mandible	Incision of oral abscess, believed to be of local origin	Infection and necrosis became severe and extensive	Leukemia caused oral abscess which was treated as local condition
BB Myeloid Chronic	Diagnosis of leukemia established by examination of blood	Swelling was present from dentoalveolar abscess	Extraction to treat dentoalveolar abscess	No noticeable untoward effects followed extraction	Possibly acute dentoalveolar abscess was exacerbation of chronic alveolar abscess. Lowered resistance from leukemic state may have accounted for exacerbation
CC Lymphoid Acute	No complaints of systemic disease	Not stated	Extraction	Within 2 days, submaxillary lymph nodes were bilaterally swollen and tender, purpura and anemia were present	As purpose for removing tooth was not stated, it is impossible to judge relation of extraction to leukemia
DD Lymphoid Acute	Increasing weakness	No clue given	Extraction	Excessive bleeding and necrosis of alveolar tissue	Same as above
EE Lymphoid Acute	Child was irritable; gingivitis was present	Caries, teeth erupting in malposition, gingivitis, fetor oris, swollen and tender submaxillary lymph nodes	Extraction to treat infected teeth—thought to be cause of tender submaxillary lymph nodes	Infection and necrosis became extensive and severe	Leukemia produced oral changes and swelling of submaxillary lymph nodes. Because of these changes, teeth were extracted

FF Myeloid Acute	No evidence	Bleeding gums	Scaling of teeth to treat what appeared to be simple gingivitis	Bleeding became severe and continuous	Bleeding gums was manifesta- tion of leukemia
GG Myeloid Chronic	Pain in lumbar region	Not stated	Extraction to remove foci of infection as measure in treating arthralgia	Not stated	Leukemic infiltration of ver- tebrae produced pain in lum- bar region. Arthralgia was possible reason for removing foci of infection
HH Monocytic Acute	Felt "below par," had not recovered from "flu," in rea- sonable length of time	Teeth that were ex- tracted were thought to be infected	Extraction. Patient's teeth were known to be "poor." Extrac- tion was carried out at this time because of poor general health	Bleeding, throbbing, and ach- ing of jaw	Teeth were removed in hope of improving general health of patient
JJ Monocytic Acute	Felt well	Teeth were neglected— oral abscess was opened. Soon 2 teeth were extracted on same side as abscess	Extraction. Alveolar abscesses	Tonsil on same side as abscess became swollen; on basis of biopsy diagnosis of lympho- epithelioma was made	Four lesions, all on right side of mouth and pharynx, were manifestations of leukemia
KK Leuco- sarcoma Acute	Arthralgia of both hips	Not stated. Physician ordered teeth extracted	Extraction. Physician's advice to have teeth removed implies that teeth were in question- able condition	Not stated. Weakness became more marked; hemorrhage into gastrointestinal tract; high fever	Possibly manifestations of leu- kemia produced arthralgia because of which teeth were extracted

TABLE VII. STUDIED CASES ARRANGED AS TO TYPE AND COMPARED WITH THE TOTAL NUMBER OF CASES OF EACH PARTICULAR TYPE

TYPE	NUMBER OF CASES OF THIS TYPE FOUND IN 111 AUTOPSIED CASES	NUMBER THAT WERE PERTINENT	PERCENTAGE OF TOTAL THAT PERTI- NENT CASES CONSTITUTE
Monocytic	8	2	25.0%
Myeloid	53	4	7.3%
Lymphoid	58	4	7.0%

indicated, there was evidence of leukemia. In 12 collected reports there was either no indication that leukemia was present before extraction, or there was no statement as to the patient's health previous to extraction. That 11 of the 12 reports are found in Table III is noteworthy. The position of the 11 reports in which there is no indication as to the presence or absence of leukemia before extraction is significant because the records in Table III are incomplete not only with regard to dental history, but probably also as to the evidence that leukemia was present before extraction.

Added to the positive evidence that leukemia precedes the dental manipulation in the instances in which it becomes conspicuous after extraction, is the consideration that extraction is common and leukemia is rare. It is concluded for the purpose of this thesis that dental procedures do not cause leukemia.

There is considerable evidence from the collected reports as well as from the studied cases that extraction sometimes causes leukemia to become acute in nature. The effects of extraction in the reports and studied cases are considered with the following question in mind: Was the life of the patient shortened by the procedure? There was evidence in 17 of the 33 collected reports that the leukemia became more acute following extraction. In 5 of the 10 studied cases, it appeared that the patient would have lived longer had the extraction not been performed. These cases and immediate changes charged to extraction are:

CC—Purpura, hemoglobin reduced to 75 per cent, death two months after extraction.

DD—Excessive bleeding, necrosis, death in one week.

EE—Osteomyelitis of the maxilla.

FF—Subcutaneous hemorrhage, death one month following extraction.

KK—Weakness becoming more marked, hemorrhage into the gastrointestinal tract, high fever.

As further evidence that leukemia sometimes takes a stormy course following dental procedures, Dr. Carl V. Moore has kindly permitted me to present the following case which has been selected from his files.

CASE LL.—The patient was a 39-year-old white housewife. A diagnosis of lymphoid leukemia was made and radiation therapy was started in April, 1943. Except for a severe sore throat and mild diarrhea, the patient was comfortable and active in her housework all of the following summer. About Sept. 5, 1943, she had a feeling of "stuffiness" in the maxillary sinuses and a clear nasal discharge. At about the time of the sinusitis, the platelet count dropped, and petechiae and purpura were present. There was improvement of the upper respiratory infection, and the patient was ambulatory although weaker than she had been previous to the sinusitis. On Oct. 7, 1943, a swelling of the lower jaw on the left side appeared. Sulfathiazole and sulfadiazine were administered, but probably



in quantities insufficient to produce a blood level of over 3 mg. per cent. On October 8, a dental abscess was opened. Evacuating the abscess cavity relieved the patient of local pain, but severe oral infection, necrosis, and hemorrhage followed the procedure within twenty-four hours. The patient's temperature increased, and signs of bronchopneumonia appeared. Fusiform and spirillar organisms compatible with those associated with ulceromembranous stomatitis were found in the sputum. The patient's course continued downhill and death occurred six days after the incision of the dental abscess.

At autopsy the pertinent findings were: lymphoid leukemia involving the viscera, stomatitis, and firm, bulging, dark red areas in the lower lobes of the right and the left lungs. The fusiform organism cultured from the sputum was not isolated from the lungs.

A discussion of the case must point out that while the occurrence and incision of an alveolar abscess marked the beginning of a precipitous downhill course, the leukemia was becoming more severe before the dental infection appeared. The appearance of petechiae and purpura, with a decrease in platelets, marked a turn in the course of the leukemia. The possibility that both the sinusitis and the dental abscess occurred because of the leukemia must be considered.

Evidence has been presented that one relation that extraction bears to leukemia is to cause the disease to become acute in its manifestations, and in some instances hasten the death of the patient. Extraction may, however, be incidental and unrelated to the leukemia. Dental procedures were associated with, but appeared to bear no relation to, leukemia in Reports 9, 18, and perhaps 32. In these reports the diagnosis of leukemia was established at the time of the dental operation, and there appeared to be no unusual response of the patient to the extraction. Case BB may represent a dental operation performed on a patient with leukemia but bearing no relation to the systemic disease. It was felt, after studying the case, that the acute abscess may have become exacerbated because of the leukemic state of the patient. The extraction was performed, of course, because of the acuteness of the abscess. If, therefore, the exacerbation of the abscess was not related to the leukemia, the extraction likewise must have been unrelated to the systemic disease.

In summary, there is no convincing evidence that extraction is a cause of leukemia. In about four-fifths of the cases and reports studied, leukemia, which was present before extraction, became more severe following this procedure. Dental operations performed during leukemia sometimes are unrelated to the leukemia.

3. *Relation Between Leukemia and Dental Procedures.*—The third part of the discussion deals with the changes produced by leukemia which may cause dental operations to be performed. It seemed from the literature and collected reports that four changes in the area about the mouth and, in addition, other general changes of leukemia were sometimes responsible for the extraction of teeth. The leukemic changes about the mouth were: (1) Swelling of the lymph nodes draining the teeth and contiguous structures. (2) Gingivitis and changes of the periodontal membrane. (3) Probably exacerbation of chronic dental abscesses. (4) Probably toothache caused by infiltration of the dental pulp by leucocytes.

The general changes of leukemia which sometimes were responsible for dental procedure were: arthralgia, loss of weight, loss of strength, and malaise. Local and general manifestations of leukemia usually did not occur separately, but often two or three were together in the reports, or in the cases studied. In four

of the studied cases dental procedures probably were performed because of oral manifestations of leukemia. The numbers of these cases and the reasons for performing the procedure follow:

<i>Case</i>	<i>Reason for Performing Dental Procedure</i>
AA	Oral abscess
EE	Swollen and painful submaxillary lymph nodes. Gingivitis with fetor oris
FF	Sore and slightly bleeding gums
JJ	Abscess over a molar tooth

In 3 of the studied cases, the procedure seems to have been performed in an attempt to relieve a systemic complaint of leukemia. The three cases and the reason for the procedure follow:

GG—Arthralgia.
HH—"Below par" feeling.
KK—Pain in hips, loss of strength.

In these 7 of the 10 studied cases, therefore, dental procedures probably were performed because of signs of acute and chronic leukemia. There was evidence in 17 of the 34 collected reports that leukemia became more acute following extraction. The finding that in 70 per cent of the studied cases dental treatment probably was given because of signs of leukemia is in agreement with the analysis of the collected reports. In 14, or 82 per cent of the 16 reports, it was thought that procedures were done because of manifestations of leukemia.

Two other relations of leukemia to dental procedures have suggested themselves as I have worked on this problem. The first of these relations is that the leukemic state is favorable to the exacerbation of chronic dentoalveolar abscesses. Many individuals have subclinical chronic dentoalveolar abscesses. It is a common clinical experience that following an "epidemic" of influenza quite a number of persons with neglected teeth complain of exacerbation of dental abscesses. Activity of chronic infection becoming active is best explained on the basis of lowered resistance. As resistance to bacterial invasion is lowered in leukemia, it seems logical to expect recrudescence of chronic abscesses. While lowered resistance may have caused the oral abscess in Case BB, and Reports 2, 5, 12, and 27, evidence was not found which would clarify the deducted upset in balance between invader and host.

The second of the deducted relations of leukemia to dental procedures rests upon changes of the pulp in leukemia. Only the observation of Prinz and Greenbaum that the dental pulp underwent "liquefaction necrosis" could be found concerning the changes of the dental pulp in leukemia. The pulp is histologically a capillary bed, and therefore it would be expected that in leukemia it would undergo the engorgement with leucocytes of other capillary beds. The dental pulp is encased in the unyielding pulp canal and chamber. Any tendency of the capillaries to expand would produce pain indistinguishable from that of ordinary pulpitis or toothache. While Forkner remarked that patients with acute leukemia complained of toothache, two opinions to the contrary were cited. Examination of a number of dental pulps from patients with leukemia reveals consistent changes and strengthens the hypothesis that infiltration of the dental pulp causes the tooth to ache.

Forkner, as well as others, stated that many patients with acute leukemia consult a dentist before a physician. In the collected reports as well as in the studied cases, patients with chronic and acute leukemia often consulted the dentist before the physician. While it was not clear in some reports whether the dentist or the physician was first consulted, it seemed that in the following reports of acute leukemia the dentist was consulted first: 1, 2, 3, 4, 5, 7, 11, 12, 13, 14, 15, 21, 25, 26, 28, 29, 31, 32, 33, and 27. (In Report 27 it was difficult to determine whether the disease was acute or chronic.) In the following reports of chronic leukemia it was likely that the dentist was consulted first: 8, 16, 17, 23, 24, and 34. In six cases of acute leukemia: CC, DD, EE, FF, HH, and JJ, and in two cases of chronic leukemia, AA and GG, the dentist first treated the patient.

#### CASE REPORTS

**CASE AA.**—Slight weakness was the first symptom of this 22-year-old man's illness. The patient was a cornetist. About a year after the onset of the weakness, there was loosening of the lower incisor teeth. A few days later, an oral abscess below these teeth was incised. Necrosis and infection of the mandible progressed. Shortly after the appearance of the oral abscess, there were 96,000 leukocytes per cubic millimeter of blood. Hemoglobin was 30 per cent. The necrosis and osteomyelitis of the mandible became more extensive. The patient's course was marked by a high and septic type of fever, and death occurred thirteen months after the loosening of the teeth had been noted.

The pertinent findings at autopsy were infiltration of the organs by myelocytes and numerous mitotic figures in the granulocytes.

*Discussion.*—The first manifestation of lymphocytic leukemia in this case was probably weakness. The record suggested that the necrosis of the mandible and loosening of the teeth were caused by phosphorus fumes around which the patient had worked. The effect of the fumes was discredited because they were from a salt and not yellow phosphorus, and because the patient was weak before he worked around the fumes. The periodontal infection, necrosis of the mandible and loosening of the teeth, and the oral abscess were most likely results of leukemia. These conditions were unwittingly treated as a local condition. The patient's avocation of playing the cornet was possibly a predisposing factor in causing the loosening of the lower anterior teeth.

**CASE BB.**—Furuncles marked the onset of this 56-year-old man's illness. A tentative diagnosis of leukemia was made before the patient was admitted to the hospital. Swelling opposite the mandible arose from an abscess of the remaining root of a lower premolar, and this root was extracted. There was no record that this chronic abscess had produced previous symptoms. With no noticeable effects from the extraction of the tooth, the patient continued to lose ground and died ten months after admission.

The pertinent findings at autopsy included the occupation of the left side of the abdomen by the spleen which weighed 1,360 grams. The liver and kidneys were large, weighing 2,080 and 355 grams, respectively. The bone marrow of the ribs appeared dirty gray in color. Microscopically, there was a general infiltration of the internal organs by cells of the myeloblastic series. The bone marrow was cellular, containing megakaryocytes in about the proportion seen in normal red marrow.

*Discussion.*—The extraction of the root was performed in order to relieve an alveolar abscess and with the realization that the patient had leukemia. The abscess was evidently a chronic infection of long standing, which had become active at this time. It is a frequent observation that intercurrent infection occurs in leukemia. It is possible that the exacerbation occurred because the patient had leukemia. This may be an example of a chronic dental infection becoming acute because leukemia lowered the patient's resistance.

**CASE CC.**—This 17-year-old boy had no complaints suggesting systemic disease before a first molar tooth was extracted. Within two days the submaxillary lymph nodes were bilaterally swollen and tender. "Shortly" after the tooth was extracted, reddish-blue

spots, which lasted about three weeks, appeared over the body. Seven days after extraction, the hemoglobin was below 75 per cent, and there was a marked necrosis of the gingiva and hemorrhage from the alveolus of the extracted tooth. The manifestations of leukemia increased in severity. The patient died two months after the extraction.

The pertinent finding at autopsy was infiltration of the organs by cells of the lymphocytic series.

*Discussion.*—Bilateral enlargement of the submaxillary lymph nodes, as a part of the general enlargement of lymph nodes, is common in lymphocytic leukemia, but extraction of a single tooth does not result in this manifestation. Similarly, purpura occurs in leukemia, but does not follow dental extraction. In all probability, the leukemia was present before extraction. While the reason for extraction is not stated, it is possible that chronic periapical inflammation became acute as a result of leukemia.

**CASE DD.**—This 66-year-old man's complaint was increasing weakness of four months' duration. A diagnosis of late syphilis was established. A lower right molar was extracted, the reason for this procedure not being stated. Following extraction, there was excessive bleeding and necrosis of the alveolar tissue. The jaw was swollen and painful. A diagnosis of acute lymphatic leukemia was made. The patient died one week later.

The pertinent findings at autopsy were leukemic infiltration in the spleen, liver, and kidneys, slight enlargement of the lymph nodes, and hyperplasia of the marrow of the femur.

*Discussion.*—The weak and comatose state of the patient was most likely a manifestation of leukemia, since the changes usually due to syphilis would be insufficient to explain this rather sudden weakness. The patient probably had leukemia at the time that the tooth was removed. Because neither the condition of the tooth, nor the reason for extraction was stated, it is impossible to determine the relation of the leukemia to the extraction.

**CASE EE.**—An 11-year-old boy was brought to a dental clinic because of toothache. The findings were "caries, general, one tooth erupting in extreme malposition, fetor oris, swollen and submaxillary lymph nodes." Three teeth were extracted. Six days later the boy was taken to the hospital. The findings upon admission were marked stomatitis with evidence of an osteomyelitis of the maxilla. There were 2,000,000 erythrocytes and 96,800 leukocytes per cubic millimeter of blood. There was slight enlargement of the lymph nodes. A diagnosis of acute lymphoid leukemia was made.

The pertinent findings at autopsy were marked lymphocytic infiltration of the organs generally, and splenic infarction.

*Discussion.*—The patient in all probability had leukemia before the teeth were extracted. A dentist was consulted in an effort to relieve the symptoms produced by leukemia. Whether the teeth ached because the pulps were infiltrated with lymphoblasts as were other organs or because chronic dental infection exacerbated, is impossible to state. It is likely that the teeth were extracted because one of these factors was operative. The impression that the patient had, primarily, inflammation of dental origin was strengthened by the swelling of the submaxillary lymph nodes. This enlargement was a manifestation of leukemia.

**CASE FF.**—This 55-year-old man consulted the dentist because of sore and slightly bleeding gums. Scaling the teeth resulted in hemorrhage which was difficult to control. Areas of subcutaneous hemorrhage followed the gingival hemorrhage. Twelve days later a diagnosis of subleukemic myeloid leukemia was made, and the patient died one month after extraction.

The pertinent findings at autopsy were cerebral hemorrhage into the left lenticular nucleus and leukemic infiltration of many of the internal organs.

*Discussion.*—The presence of the bleeding gums suggests that the patient had leukemia before he consulted the dentist. The dental treatment was given hoping to relieve a condition thought to be local in nature, but actually leukemic.

**CASE GG.**—This 50-year-old man was well until about three weeks before he entered the hospital, when he complained of pain in the lumbar region. Several teeth were ex-



tracted soon after the onset of the neuralgia. The complaint of severe pain, which radiated from the right buttock down a rather narrow zone of the flexor aspect of the thigh to the knee, persisted and became more severe.

On the basis of a biopsy from the conspicuously enlarged right tonsil and the sternal marrow, a diagnosis of lymphoepithelioma of the tonsil with metastasis was made.

The pertinent finding at autopsy was myeloid leukemia, confined to the ribs, liver, pancreas, and vertebrae.

*Discussion.*—Considerable periodontal sepsis is often seen in patients of this age. It is quite possible that the teeth were extracted in the hope that the arthralgia would be relieved. The lumbar pain was probably caused by leukemic infiltration of the vertebrae.

CASE HH.—The health of this 30-year-old man had been "generally very good" until two years before his present illness. At that time he felt "below par" without any definite symptoms. While not fully recovered from what was manifestly influenza, four teeth were extracted. This was followed by bleeding, throbbing, and aching of the jaws. Fever persisted, reaching 100° to 101° F. every second or third day. The course of the disease was rapid, and the patient expired three weeks after the diagnosis of monocytic leukemia was made.

The pertinent findings at autopsy were infiltration of the organs by monocytic leucocytes and petechial hemorrhages of the pleura, peritoneum, and pericardium.

*Discussion.*—It is impossible to establish the onset of the leukemia, but it was probably the "below par" feeling.

Here is, then, an educated man of 30 years in a fairly fortunate economic situation, who, after feeling "below par" for some time, had four teeth extracted at one time. While no reason for the extraction was stated, it is doubtful that four teeth would have been extracted at one time unless some specific effort was being made to improve the general health of the man. The teeth were probably removed to relieve vague symptoms of leukemia.

CASE JJ.—At the time of extraction of a lower canine tooth, this 40-year-old man had no symptoms of systemic disease. No statement could be found as to why the extraction was performed. In less than three weeks following extraction an abscess appeared over the upper right second molar. One week following the extraction of the second molar and the incision of the alveolar abscess, the right side of the tongue became sore, and the right tonsil swollen, reddened, and painful. The patient felt that the onset of the illness occurred at the time of the first extraction. A diagnosis of monocytic leukemia was made and the patient died about five months after the first extraction.

The pertinent finding at autopsy was monocytic leukemia involving the bone marrow, axillary, cervical, and tracheobronchial lymph nodes and the abdominal viscera.

*Discussion.*—There are features of this case which indicate that the extractions were not done merely because of local disturbance. The necessity of extracting a lower canine tooth alone is infrequent in a 40-year-old person. The patient had four lesions, all on the right side of the oral cavity and oropharynx, within three months. It is quite improbable that a dentist would extract a tooth but overlook another dental condition which was to result in an abscess in less than three weeks. Closely following, or concurrent with the dental infection, there was soreness of the right side of the tongue and swelling of the right tonsil. Evidently these anatomically associated lesions were not fortuitous. It is well recognized that monocytic leukemia often first manifests itself by oral lesions. It seems that the conditions which indicated the removal of the teeth as well as the soreness of the tongue and swelling of the tonsils were manifestations of the leukemia.

CASE KK.—This 52-year-old male had pains in both hips and increasing fatigue. On the advice of his physician, all of his remaining teeth were extracted. The general condition of the teeth is not stated. Closely following the extraction of the teeth there was marked weakness, tarry stools, a mass with pain over it in the left upper part of the abdomen, and a temperature of 102° to 103° F. The patient became very weak and was

dyspneic. The total leucocytic count was 82,700. A diagnosis of leucosarcoma, type not stated, was made, and the patient died four months later.

Pertinent findings at autopsy included petechiae over all parts of the body, enlargement of the spleen and kidneys. Microscopically, small cells, from 7 to 12 micra in diameter, with hyperchromic nuclei and scant cytoplasm were found infiltrating the organs, particularly the heart, lung, liver, spleen, kidney, and duodenum. This same type of cell was found in the bone marrow.

*Discussion.*—This patient, like the one described in Case GG, was an older man with some periodontal alveolar atrophy and sepsis. In all probability the teeth were extracted hoping to relieve the pain in the hips. Since infiltration of the vertebral bone marrow produces lumbar pain, and infiltration of the kidneys may produce low back pain, the pain in the hips was possibly a manifestation of leukemia.

#### CONCLUSIONS

1. A relation exists between dental procedures and leukemia in nearly 1 case in 12 of leukemia.
2. The incidence of a relation between procedures and leukemia is strikingly high when the monocytic type is considered.
3. The relation between dental operations and the systemic disease has two aspects:
  - a. The leukemia becomes more acute in approximately half of the patients who undergo dental operations. Oral lesions nearly always become more conspicuous and sometimes destructive following extraction in acute leukemia. Stomatitis often follows extraction in chronic leukemia. Dental operations performed on patients with leukemia may, however, be unrelated to the systemic disease.
  - b. Because signs of leukemia are sometimes misinterpreted as being of dental origin, dental procedures are performed. Of 16 reports in which the reason for performing the procedure was clear, in 14, and also in 7 of the studied cases, procedures seemed to have been performed because of the signs and symptoms of leukemia. Patients with early leukemia often consult the dentist before the physician.
4. Two possible relations of leukemia to dental disease are suggested. (a) Lowered resistance to bacterial invasion because of leukemia may become manifest by recrudescence of chronic dental abscesses. (b) Leukocytic infiltration of the dental pulp may cause toothache in leukemic patients.

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## THE USE OF FIXED AND REMOVABLE SPLINTS IN THE PRACTICE OF PERIODONTIA

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FOR many years, the writer has advised the use of stationary and removable splints as aids in the healing of the supporting tissues about mobile teeth. Temporary splinting often aids to such an extent that after a short period of time the device may be discarded, the teeth becoming so firm that they are able to assume their normal functional load. At other times the construction of a permanent splint becomes necessary.

With the introduction of acrylic (methyl methacrylate) into dentistry, a new material for temporary splinting of loose teeth may now be employed in the practice of both conservative and radical procedures of periodontal treatment. The material has so many advantages that it may supplant other materials which have been previously employed.

Removable splints may be employed in the following conditions:

1. When teeth are displaced or loosened by accident.
2. To support loose teeth from excessive injury.
3. To stabilize a tooth suffering with pericementitis, so that it can be ground to relieve occlusal pressure, or while procedures are undertaken to eliminate the cause.
4. To stabilize the teeth until a permanent splint is constructed.
5. To stabilize teeth during gingivectomy.
  - a. The splint keeps the sedative pack in position.
  - b. It prevents shifting of teeth while healing takes place.
6. To prevent cheek-biting.

The materials most commonly used for temporary splinting are: (1) thread (grassline, silk, linen, etc.), (2) wire (gold, steel, brass), (3) cast or wrought metal (steel, chromium alloys, gold).

Although these substances have been effective, many disadvantages have been encountered.

### USE OF THREAD

The usual procedure in using thread is to select two firm teeth which will serve as anchorage. A clove hitch is slipped over the first stable tooth and secured with a knot (Fig. 1, *A* and *B*.) The ligature is then fastened around each mobile tooth, knotting in each interdental space, the thread being kept above the cingulum. This is continued until the other firm tooth is reached, whereupon a surgeon's knot is tied on the distal surface. If large interdental spaces are encountered, a sufficient number of knots are tied interdentally to fill the spaces,

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to avoid traction (Fig. 2). Other methods in employing thread are used for the same purpose. Disadvantages of thread splinting are: (1) The teeth are not completely stabilized. (2) Traction may result. (3) The ligature may slip and cause teeth to move out of line. (4) It interferes with toothbrush stimulation. (5) It may irritate lip, tongue, or cheek. (6) It is difficult to use in the posterior portion of the mouth.

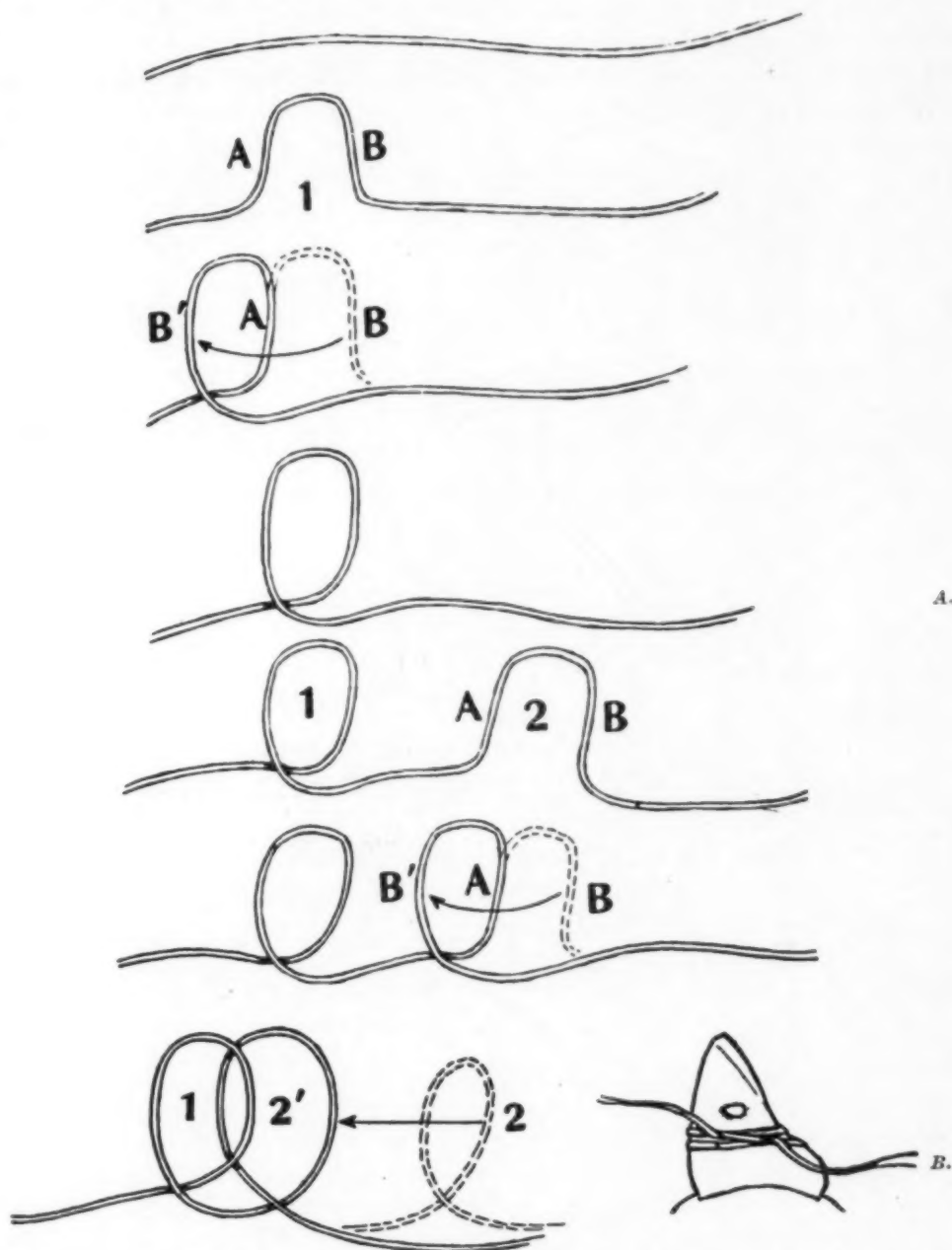


Fig. 1.—A, Technique for making the clove hitch for tying the silk or grassline ligature to the first tooth.

B, Double loop completed. Note that the strand of loop 1 is under the loop while the strand of loop 2 is over it. Loop 2 is now placed under loop 1.

Silk or grassline ligature on first tooth continues the splint.

(From Miller: Textbook of Periodontia, 1943, The Blakiston Co.)

## USE OF WIRE

Stainless steel wire is used most universally today for wiring of teeth. A number of methods are employed, the most popular one of which was described in 1915 by Dr. Robin Adair, who credits Dr. Spies as the designer. Recently, other men have described this method, not realizing that the method had been published previously.

A slip noose of wire (brass, gold, steel), 26 gauge, is placed around all the teeth to be splinted (Fig. 3, *cc*). Thus, if the teeth splinted cover the area from cuspid to cuspid, the wire is placed between cuspid and premolar on one side and between the same teeth on the other side. It is kept above the gingulum



Fig. 2.—Silk or grassline ligature in position. Note double loop on first tooth (first premolar) and multiple knots where space is present between teeth (indicated by arrows). (From Miller: Textbook of Periodontia, 1943, The Blakiston Co.)

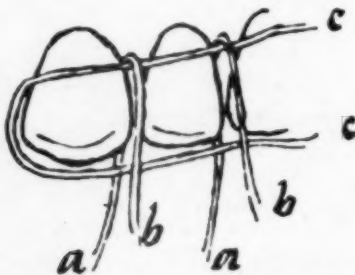


Fig. 3.—Method of ligating teeth with brass or stainless steel ligature. *cc* is a 26 gauge ligature passed around all teeth to be splinted; *ab* is a 30 gauge ligature which is tied at each interproximal space involved. (From Miller: Textbook of Periodontia, 1943, The Blakiston Co.)

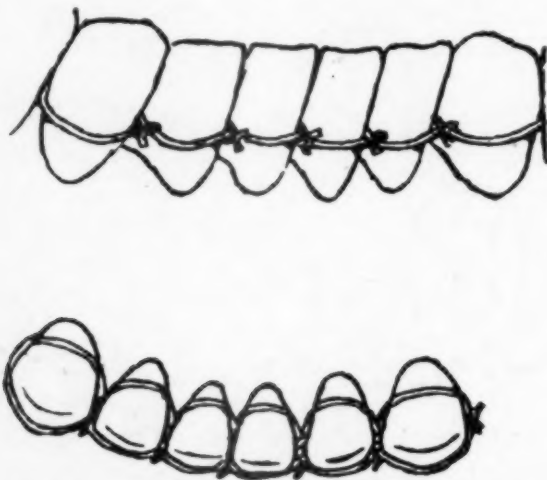


Fig. 4.—Facial and incisal view of teeth with brass or stainless steel splint showing how the knots are tucked into the embrasures to avoid irritation to the lips or tongue. (From Miller: Textbook of Periodontia, 1943, The Blakiston Co.)

and tied loosely at one end. A thinner (30) gauge wire (Fig. 3, *ab*) is now introduced into each interdental space, one strand going above the slip noose, the other going below. The wire is twisted until the teeth are firm. Each wire is cut after twisting and then turned back interdentally to prevent irritation to the lip, tongue, or cheek. After all the interdental wires are in position, the slip-noose wire is tightened as desired. (Fig. 4.) Other methods employing wire (J. A. Jungman Lacing Method) have been utilized.

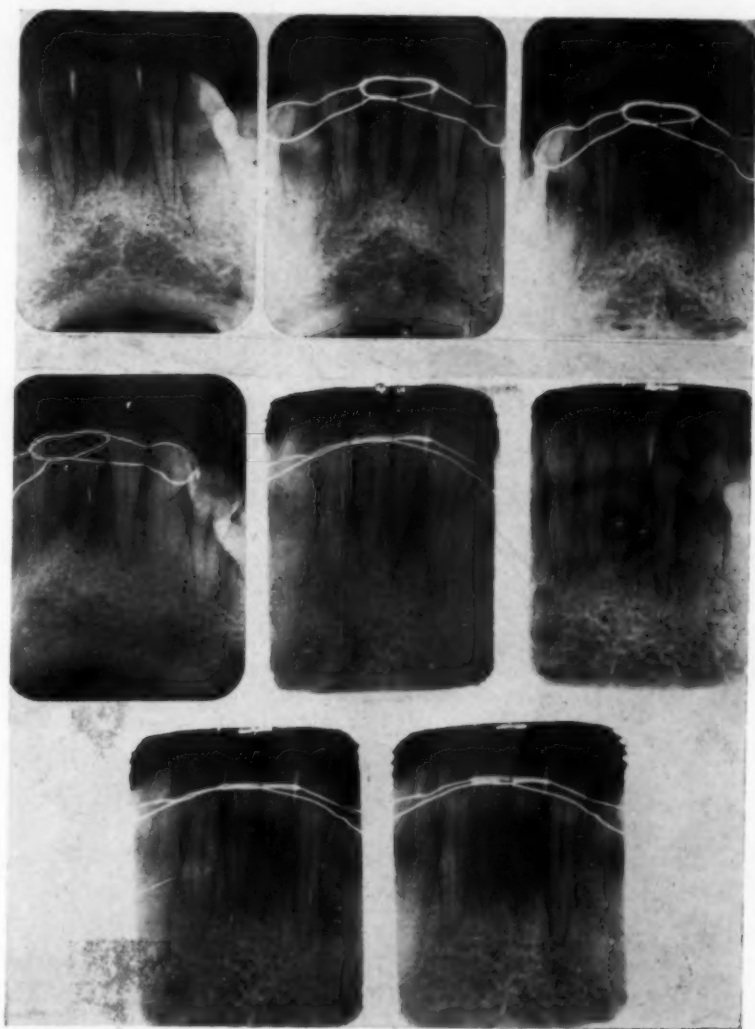


Fig. 5.—Use of wire in the splinting of teeth. Note bone filling in due to stabilization. (From Miller: *Textbook of Periodontia*, 1943, The Blakiston Co.)

The disadvantages of wire are: (1) It does not completely stabilize the teeth. (2) It is unsightly. (3) It is difficult to control the pressure applied between teeth. (4) Wires require continued adjustments (Fig. 5). (5) Lips, cheeks, and tongue may become irritated. (6) It interferes with brushing.

#### USE OF METAL SPLINTS

There are two types of metal splints:

1. The band technique type. Each tooth to be splinted is banded, then soldered together and cemented into place.

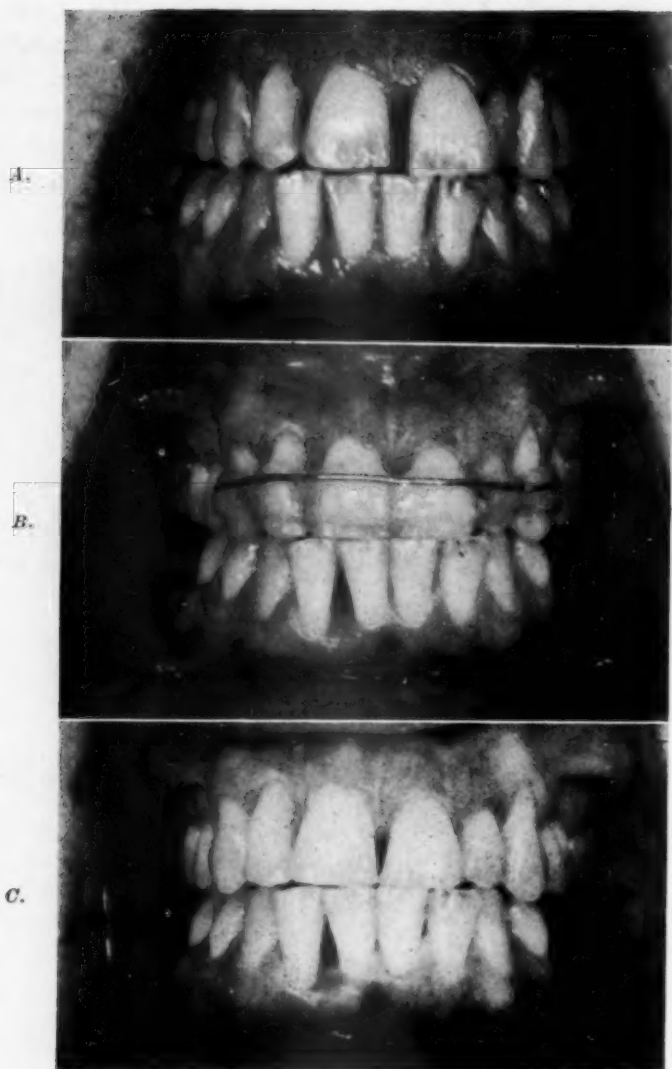


Fig. 6.—Miss F., aged 38 years. A, Before treatment. Note separation of teeth. B, Teeth brought into position and maintained by Sorrin acrylic splint. C, Six months after removal of splint. (From Miller: *Textbook of Periodontia*, 1943, The Blakiston Co.)



Fig. 7.—Sedative pack employed after gingivectomy, with Sorrin splint in position. (From Miller; *Textbook of Periodontia*, 1943, The Blakiston Co.)



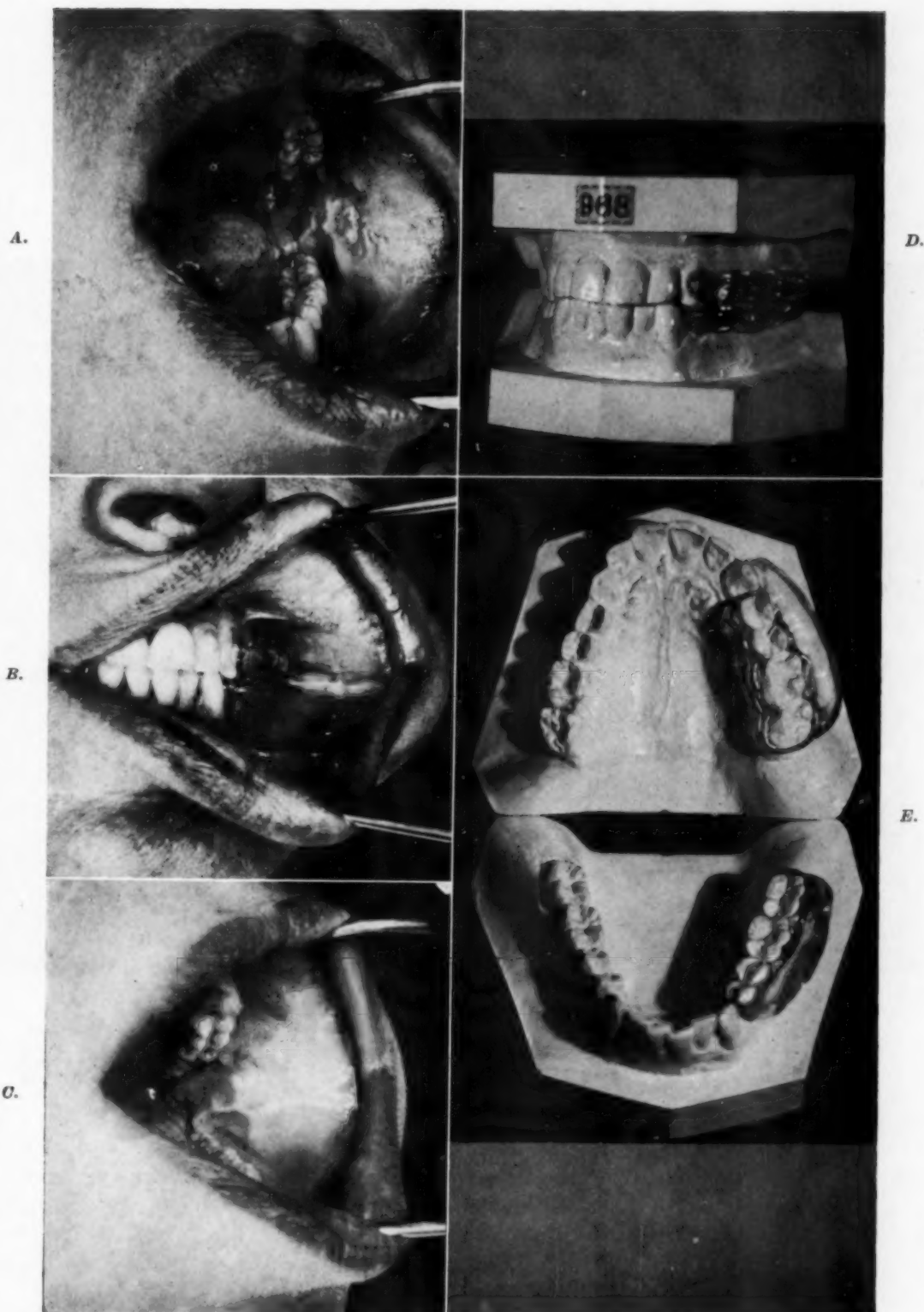


Fig. 8.—Case of cheek-biting. *A*, Note leucoplakia. *B*, Splint with buccal bumpers in position on upper and lower teeth. *C*, Completely cured. Habit ceased and leucoplakia disappeared. *D*, Model of splint with bumpers. *E*, Another view of splint with bumpers. (From Miller: Textbook of Periodontia, 1943, The Blakiston Co.)

2. The continuous clasp type. The appliance encircles the labial and lingual or buccal and lingual surfaces of the teeth at the heights of contour and is placed into position. If many teeth are to be included in this splint, the buccal and lingual portions may be joined together at various regions in the mouth. Interference with occlusion must be carefully checked.

The disadvantages of the metal splints are: (1) Unsightly appearance. (2) Metallic taste. (3) Difficult to use where teeth are out of line.

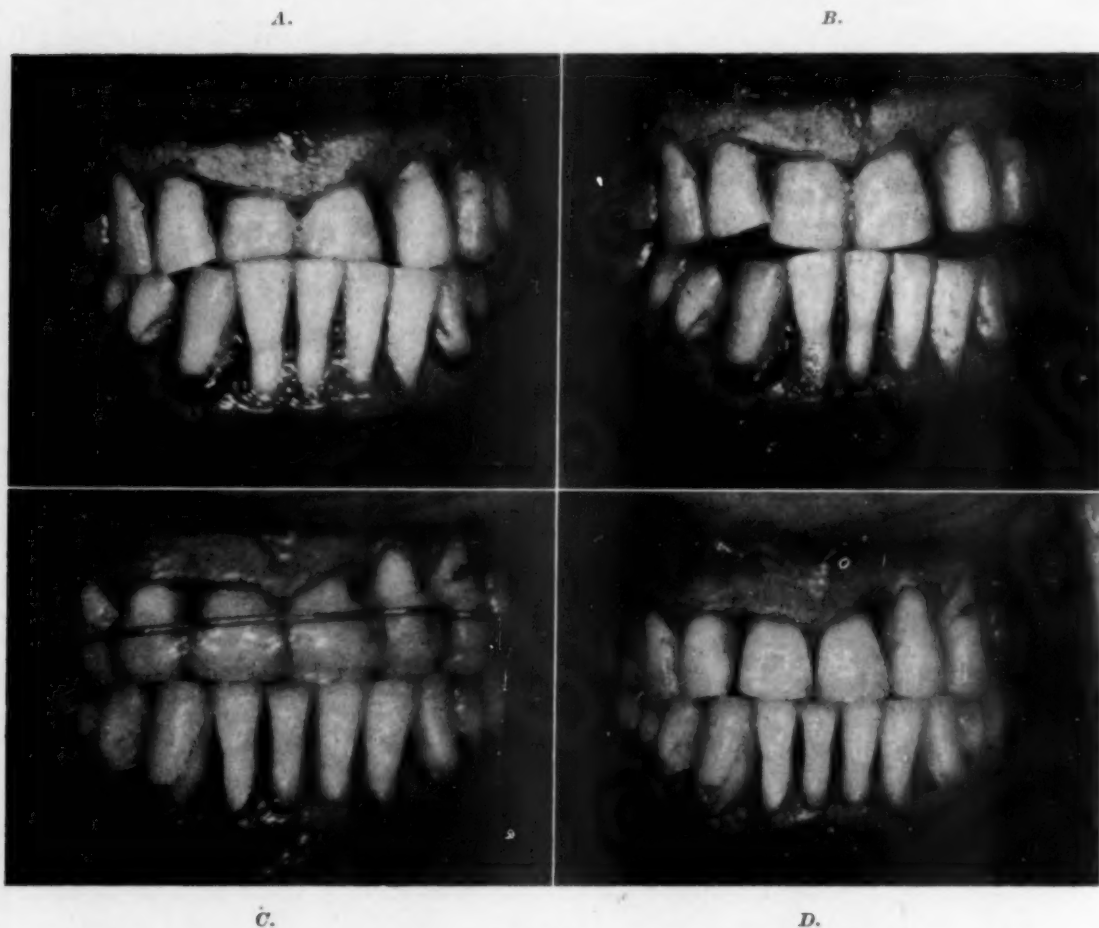


Fig. 9.—Man, aged 40 years. *A*, Prognathous jaw. *B*, Protrusive position. Mouth is ground into a tip-to-tip relationship and anterior teeth are ground until posterior contact is obtained. *C*, The upper anterior teeth have moved into better position and are maintained by acrylic splint. *D*, Six months after removal of splint. Note great improvement. (From Miller: Textbook of Periodontia, 1943, The Blakiston Co.)

#### USE OF SORRIN ACRYLIC SPLINT

This removable splint may be employed in both the conservative and more radical methods of treatment. Before gingivectomy is attempted, the acrylic splint, reinforced with wire, is constructed and placed into position. Its use is best confined to small segments. (I am using this splint most exclusively since I introduced it in 1942.) (Figs. 6, 7, 8, 9, and 10.)

#### METHOD OF CONSTRUCTION

1. Take impression in plaster, hydrocolloid, or alginate material.
2. Make a stone model.

3. Contour stainless steel wire (21 gauge) wherever desired. In a splint for anterior teeth, the wire is contoured on the lingual surfaces above the gingulum. Carry the ends of the wire around the distal and labial surfaces of the cuspid teeth. (Fig. 6.) Remove the wire.

4. Tin foil the model with 0.001 foil.

5. Place the wire over the tin foil and wax up labial and lingual surfaces. Do not wax around wire at the point where it passes from lingual to labial surface distal to the cuspid.

6. Adapt 0.001 tin foil over the exposed wax.

7. Embed the model in the lower half of the flask. Use plaster.



A.



B.



C.

Fig. 10.—Woman, aged 37 years. A, Note shifting of teeth. B, After moving of teeth into position, teeth were maintained by acrylic splint. C, Six months after removal of splint on upper teeth. (From Miller: *Textbook of Periodontia*, 1943, The Blakiston Co.)

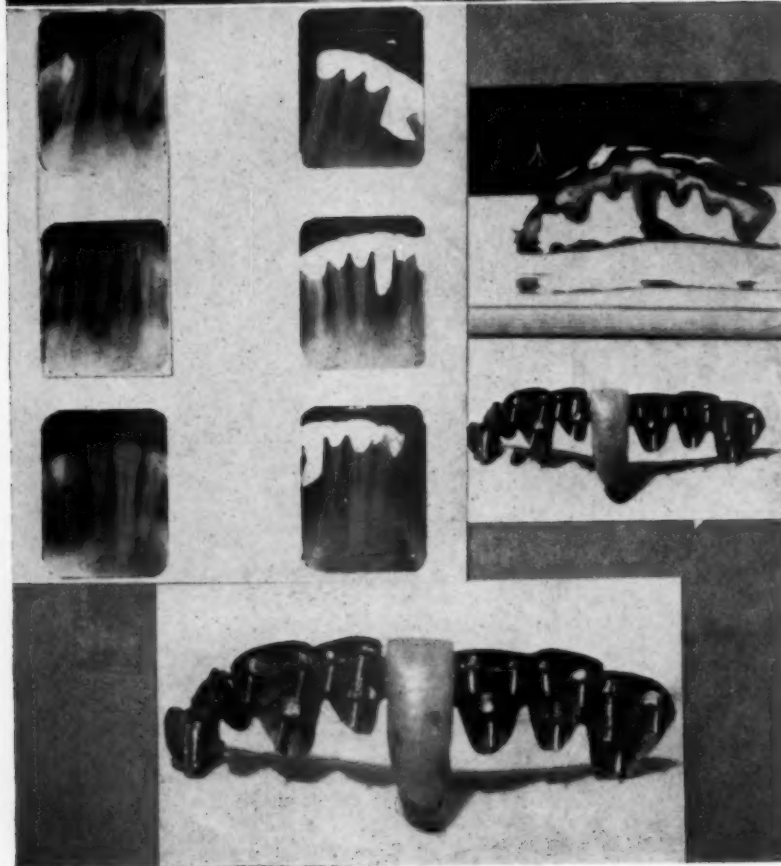
A.



B.



C.



E.

D.

Fig. 11. (See opposite page for legend.)

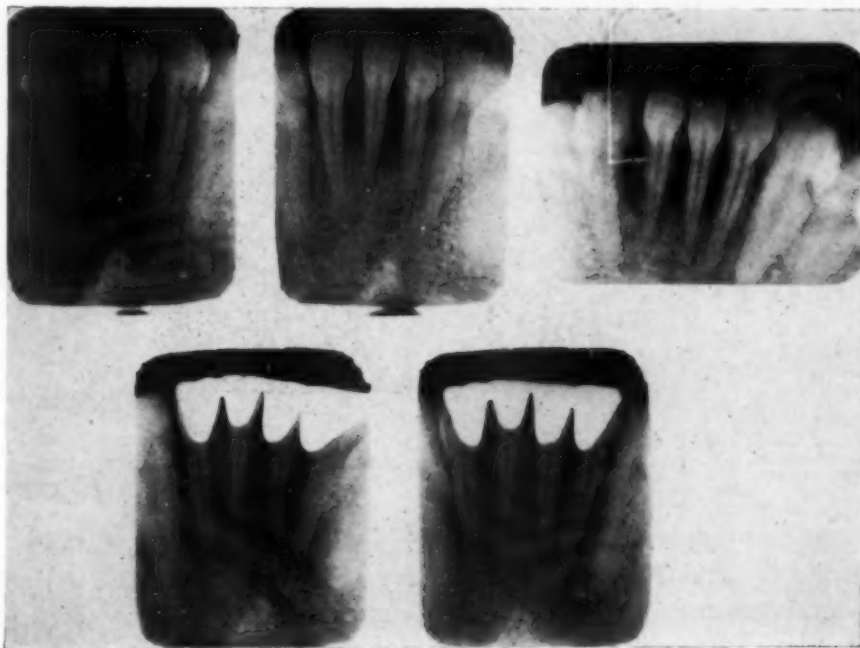


8. Coat investment with separating medium, and pour upper half of flask.
9. After the investment has set, separate the flask and flush with boiling water, making certain that all wax and coloring matter are removed.
10. Use clear acrylic and process in the usual manner.

## ADVANTAGES OF THE ACRYLIC SPLINT

1. It mobilizes the teeth.
2. It is smooth to the tongue, lips, and cheeks.
3. It has an almost perfect adaptability for enamel. Its slight flexibility and festooning hold the teeth securely, requiring no adjustments.
4. It is unaffected by the saliva.
5. It is not unsightly. The clear acrylic allows the natural tooth to be observed.
6. It is not displaced during the use of the toothbrush.
7. It may be employed in cases where teeth have been moved into position and require maintenance (Fig. 6, *A*, *B*, and *C*).
8. It keeps the sedative packing in position (Fig. 7).
9. It may be employed to prevent cheek-biting (Fig. 8, *A*, *B*, *C*, *D*, and *E*).
10. It is used while preparing teeth for mouth rehabilitation.
11. It may be used on anterior and posterior teeth.

A.



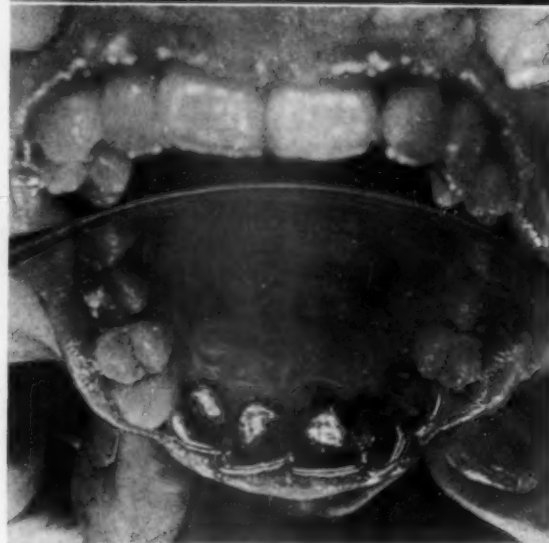
B.

Fig. 12.—*A*, Case before treatment, October, 1934. Pinledge placed into position, April, 1935. *B*, Ten years later. Note bone regeneration. (From Miller: *Textbook of Periodontia*, 1943, The Blakiston Co.)

Fig. 11.—Woman, aged 35 years. This case required splinting to be better enabled to support a partial denture. *A*, Before insertion of pinledge. *B*, Pinledge in position. *C*, (*a*) Roentgenographs before insertion of pinledge, (*b*) Roentgenographs with pinledge in position. *D*, Enlarged view of Burgess pinledge splint. *E*, Lingual view of pinledge through a mirror. (From Miller: *Textbook of Periodontia*, 1943, The Blakiston Co.)

The disadvantage of acrylic is its weakness when applying too much pressure. While not as strong as metal, this is overcome by reinforcing with stainless steel wire. Where esthetics demand, the wire can be eliminated, but little pressure should be used to seat such a splint. If too much pressure is employed, the acrylic may break.

A.



B.

Fig. 13.—A, Labial view with cast pinledge of upper anterior teeth in position in 1937. B, Lingual view of cast pinledge in 1944. (From Miller: Textbook of Periodontia, 1943, The Blakiston Co.)

#### SUMMARY

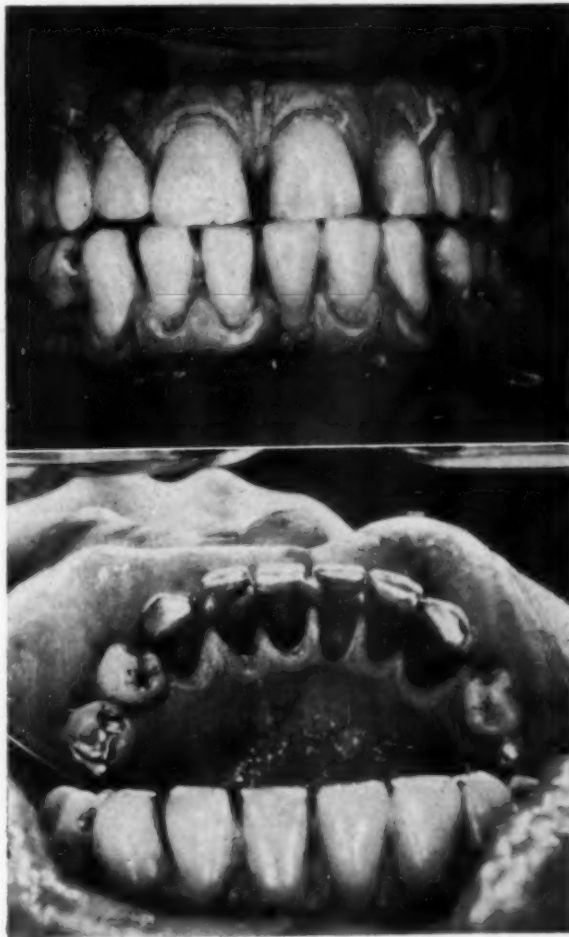
If the removable splints are worn for a considerable period of time, and periodontal treatment is carefully instituted, there may be a return to health of the supporting tissues, thus avoiding the necessity of permanent splinting (Fig. 9, A, B, C, and D, and Fig. 10, A, B, and C).

#### FIXED OR STATIONARY SPLINTS

Fixed or stationary splints have proved to be excellent aids in the retention of natural teeth which otherwise would have been extracted. One of the pioneers in this type of dentistry was James Kendall Burgess, who introduced the

pinledge splint over twenty years ago. It was a serviceable aid in restoring weakened teeth to firmness and even to this day is still serving a useful purpose. (Fig. 11.) In this case the splint is still serving in the mouth after fifteen years. The pins were soldered to gold foil which had previously been contoured to the preparations. Then all individual pinledges were placed into position and soldered together. Another such case is observed in Figs. 12 and 13. In recent years, a number of new developments have occurred: the casting of pinledge bridges (Figs. 14, 15, and 16), and the introduction of pit-veneer and veneer three-quarter attachments.

A.



B.

Fig. 14.—A, Labial view with cast pinledge in position in 1942. B, Lingual view of the lower anterior splint in 1944. (From Miller: *Textbook of Periodontia*, 1943, The Blakiston Co.)

#### PIT-VENEER AND VENEER THREE-QUARTER ATTACHMENTS\*

Teeth under stress are known to have three different and distinct types of movement that must be taken into account when constructing a splinting appliance: (1) buccolingual, (2) vertical, and (3) rotational.

Because the incisal edge is not covered and the proximal surfaces are not gripped by a pinledge attachment, the forces of mastication may eventually actually unseat the tooth from

\*Miller, S. C.: *Textbook of Periodontia*, ed. 2, Philadelphia, 1943, The Blakiston Co.

the casting. Another factor, not taken into consideration in the construction of splints employing pinledge attachments, is extension for prevention of caries. Because the proximal margins do not extend into cleansable areas and, because, in a mouth requiring a splint, hygiene is of still greater importance than in a healthy mouth, the pinledge should be considered only academically in the evolution of modern fixed splints, and its use avoided entirely at present.

In the Crown and Bridge Department of New York University College of Dentistry, a technique different from those in common use has been developed. Both the veneer three-quarter crown and the pit-veneer attachment are employed. In the preparation of the pit-veneer, the proximal wall adjacent to the next tooth to be attached is included through the contact point to bring the labial margin out into a cleansable area, thus following the formula of extension for prevention. When pit-veneer attachments are used, they are constructed for

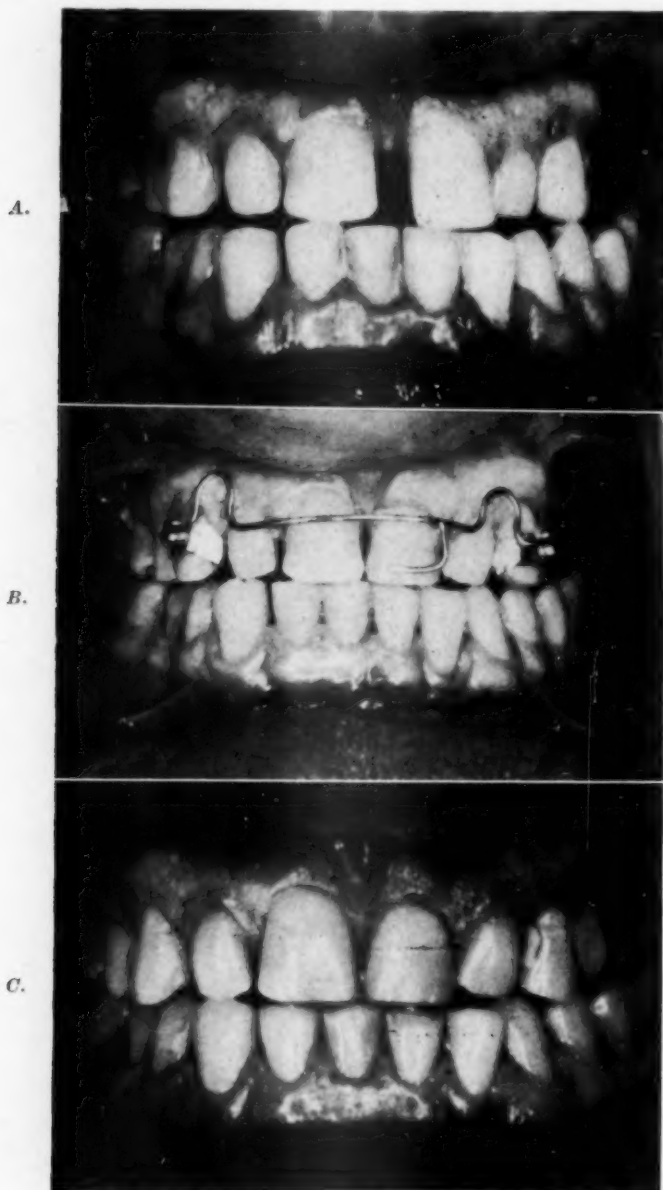


Fig. 15.—Case of Mrs. A., aged 38 years. A, Teeth separation. B, Teeth brought into position by simple orthodontic appliance. C, Pinledge splint in position. (From Miller: *Text-book of Periodontia*, 1943, The Blakiston Co.)



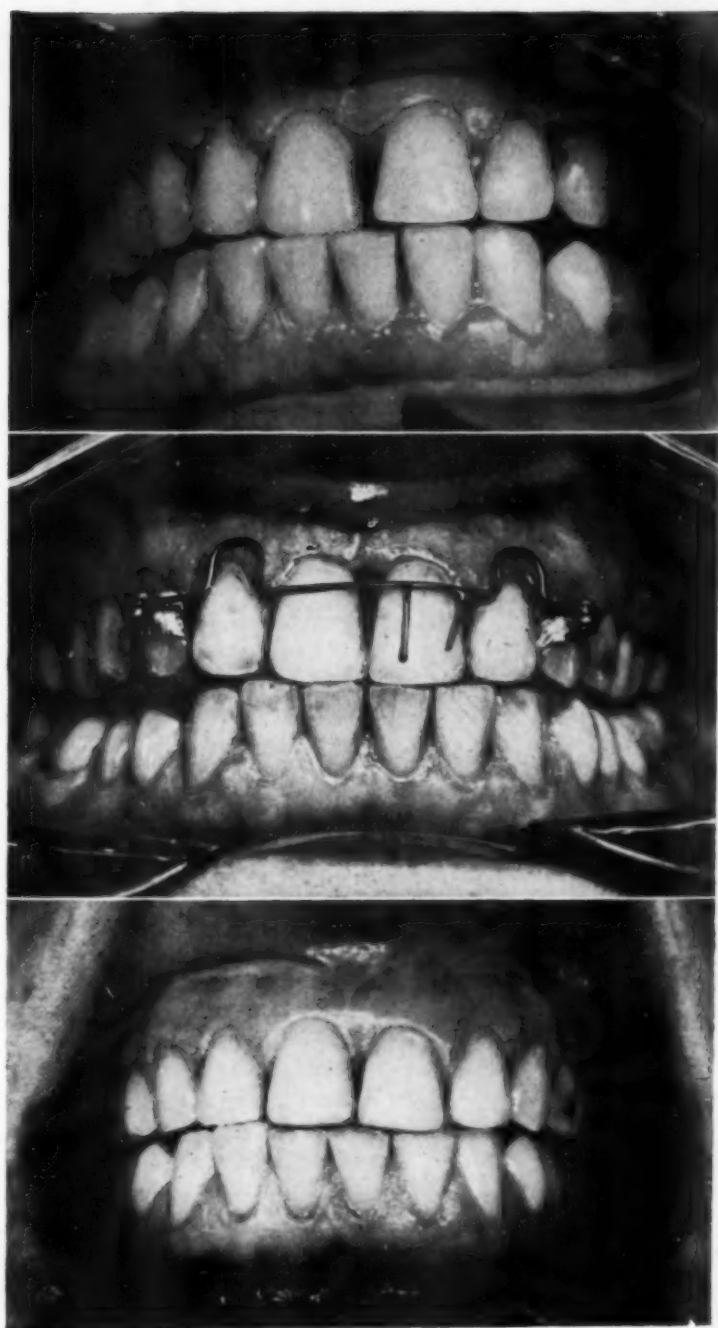


Fig. 16.—Woman, 40 years of age. *A*, Separation between teeth of upper jaw. Note also separation between teeth of lower jaw. *B*, Orthodontic splint which was helpful in moving teeth into position. *C*, Note pinledge splint in position, uniting upper right central incisor, left central, and left lateral incisors. (From Miller: Textbook of Periodontia, 1943, The Blakiston Co.)

the distal anchorage, while three-quarter crowns are used on all the intermediate teeth to be included in the splint.

Since the development of the pit-veneer attachment by Kabnick, it is possible to eliminate the inclusion of the distal surfaces of the terminal abutments, thus preserving the natural contact points. It must be emphasized, however, that great care in diagnosis must be taken in selecting a tooth on which to construct a pit-veneer attachment, for there are more contraindications for the use of this preparation than there are indications.

The crown must be of sufficient length to allow for a large slice on the proximal surface in which to place a substantially long locked retention groove. Moreover, the buccolingual thickness of the crown must be sufficient to allow, first, that the groove be locked without undermining the labial plate of enamel, and second, that the anchorage pits be sufficiently long for adequate retention without endangering the pulp or showing through the labial plate of enamel.

#### PREPARATION OF TOOTH FOR THE PIT-VENEER

With a safe-sided disk, cut through the surface of the tooth which approximates the next member which is to be included in the splint, thus establishing the soldering surface. This slice is cut through the contact point, holding the disk at a 30 degree angle. With a No. 4 Crystalon stone, an incisal bevel (Fig. 17, *C*) of 1 to 2 mm. is made, care being exercised to see that sufficient clearance is established during the various excursions of the mandible. Using a knife-edged stone, a groove (*b*) is cut in the lingual about 1 mm. in depth, starting about 1 mm. from the incisal enamel plate to the neck of the tooth. Now reduce the lingual surface (*c*) to two thicknesses of heavy carbon paper, with a No. 11 Crystalon stone. The initial slice now is joined up with the knife-edged groove at the base of the marginal ridge (*d*), using the No. 4 Crystalon stone. With a rounded No. 16 Crystalon stone the margins of the knifed groove (*b*) are finished off giving it a bevel. The lock groove (*e*) is started at the lingual termination of the incisal bevel (*a*) and is made parallel to the incisolabial two-thirds of the tooth. The groove is started with a Kruse or No. 800L cross-cut fissure bur, followed by a No. 700L cross-cut fissure bur sunk to its depth. Then, with a buccolingual movement of the bur in the groove, the lock is established. Both the cervical and incisal pits are made with a No. ½ round bur, sunk to 1½ to 2 mm. in depth, depending on the thickness of the tooth structure. These pits are made parallel to the lock groove or incisolabial two-thirds. The pits are now trued up with a No. 556 cross-cut fissure bur.

#### PREPARATION OF TOOTH FOR THE VENEER THREE-QUARTER CROWN

Both the mesial and distal slices are made at the expense of the proximal surfaces with a safe-sided disk held at an angle of 30 degrees (Fig. 18, *C*). The incisal bevel at an angle of 45 degrees (*b*) is made with a No. 4 Crystalon stone. The lingual reduction (*c*) is made with a No. 11 Crystalon stone permitting clearance of four thicknesses of heavy carbon paper. The mesial and distal slices, and the reduced lingual surface are joined at the cervical (*d*) with a coarse granite sandpaper disk followed by a fine disk. The lock grooves (*e*) and the pit (*f*) are made the same as in the pit-veneer construction.

In both the pit-veneer and the veneer three-quarter crowns, the wax-up is done directly in the mouth, insuring a closer adaption and producing a frictional grip of the attachment to the tooth. With a heated Kerr No. 10 root canal plugger, the wax can be forced into the pits, thus making a one-piece casting. On all teeth, the preparation is made to include the incisal edges, so that opposing teeth will occlude with a gold surface instead of the tooth structure, thus avoiding the possibility of depressing the tooth in its socket and forcing it away from its attachment.

#### STATIONARY POSTERIOR SPLINTS

Where there has been considerable loss of bone or where teeth have a tendency to drift, permanent splints are often necessary. Although the lower anterior teeth are by far the most frequently splinted, any tooth or group of teeth may be stabilized providing pathosis has been

Fig. 17.

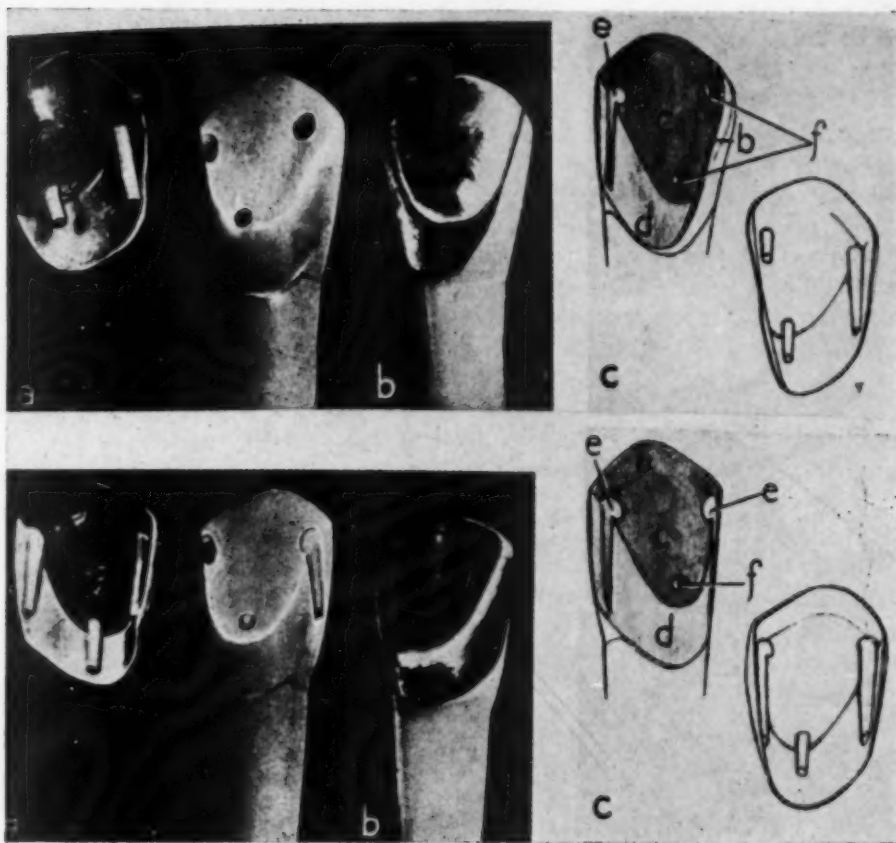


Fig. 18.

Fig. 17.—*a*, Lingual view of attachment and pit-veneer preparation. *b*, Pit-veneer attachment in place showing preservation of one proximal surface. *c*, Diagrammatic view of pit-veneer preparation and attachment. Note dovetail groove. (From Miller: *Textbook of Periodontia*, 1943, The Blakiston Co.)

Fig. 18.—*a*, Lingual view of attachment and veneer three-quarter preparation. *b*, Veneer three-quarter attachment in place. *c*, Diagrammatic view of veneer three-quarter preparation and attachment. Note dovetail grooves. (From Miller: *Textbook of Periodontia*, 1943, The Blakiston Co.)

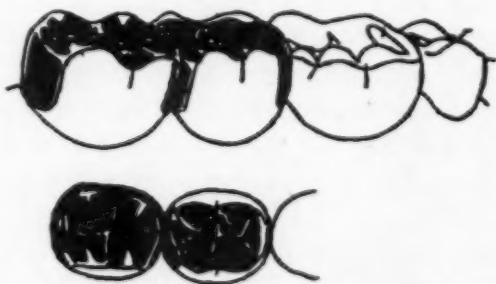


Fig. 19.—Posterior splint. It is best to use three-quarter attachments or at least mesioclusodistal inlays with the entire morsal surface covered on each tooth involved in the splint. (From Miller: *Textbook of Periodontia*, 1943, The Blakiston Co.)

eradicated and there are sufficient supporting teeth present. In the posterior region, where a third molar has been removed, it may be found that the second molar moves distally, thus opening the contact with the first molar. Here three-quarter crowns often are constructed for each of the molars in question, and their contact is soldered. In this way there can be no further movement distally of the second molar and the food impaction area is eliminated entirely. (Fig. 19.)

Whenever any tooth is splinted, whether it be anterior or posterior, the biting surface must be covered completely. By carrying the attachment over the incisal or morsal surface, the possibility of loosening the tooth from the splint is avoided. The former use of movable joint, MacBoyle, or simple mesioclusal or distoclusal inlay attachments must be condemned. The MacBoyle attachment accomplishes no more than the three-quarter crown while it creates greater difficulty in preparation and fitting and a greater number of margins for possible recurrent decay. The simple two-surface inlay does not give sufficient retention for splinting.

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745 FIFTH AVENUE

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### CLASSIFICATION OF CYSTS OF THE JAWS

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A NUMBER of classifications of oral tumors, including cysts, have appeared in the literature from time to time.<sup>9</sup> That proposed by the British Dental Association's Committee on Odontomes<sup>3</sup> is probably the most generally accepted. Among others that have been proposed by various authors are those of Bland-Sutton,<sup>2</sup> Wohl,<sup>17</sup> Churchill,<sup>4</sup> Ivy and Churchill,<sup>6</sup> Geschickter,<sup>5</sup> Bernier,<sup>1</sup> Robinson and Koch,<sup>10</sup> Thoma,<sup>15</sup> and Waldron.<sup>16</sup> Unfortunately, most of these classifications have overlooked the basic etiology of the lesions concerned, have developed such bulky indices that they have become impractical, or have included odontomes which are actually anomalies, with cysts and neoplasms.

The following classification is based on a study of development, structure, and radiographic appearance of the lesions. It is offered in an effort to simplify diagnosis for the clinician, radiographer, and pathologist.

Presented at the Twenty-Second General Meeting of the International Association for Dental Research, Chicago, Ill., March 18, 1944.

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\*The Ohio State University, Columbus, Ohio, and Washington University, St. Louis, Mo.



## CLASSIFICATION\*

- I. Developmental cysts
  - A. From odontogenetic tissue
    - 1. Periodontal cyst
      - a. Radicular or dental root apex type
      - b. Lateral type
      - c. Residual type
    - 2. Dentigerous cyst
    - 3. Primordial cyst
  - B. From nondental tissues
    - 1. Median cyst (median palatine cyst)
    - 2. Incisive canal cyst
    - 3. Globulomaxillary cyst
- II. Cystic neoplasms (dental origin)
  - 1. Ameloblastoma

For completeness or radiographic and clinical diagnosis the following lesions which may appear cystic, especially radiographically, should be considered although they are not classified as cysts:

- a. Extravasation "cysts" (traumatic and nontraumatic hemorrhagic "cysts")
- b. Neoplasms resembling cysts due to bone destruction
- c. Metabolic dysfunctions resembling cysts due to bone destruction
  - i. osteitis fibrosa cystica
  - ii. osteitis deformans (fibrotic stage)
  - iii. xanthomatoses
  - iv. eosinophilic granuloma
  - v. multiple myeloma
- d. Inflammatory disease resembling cysts due to bone destruction.

## DEFINITIONS

A cyst is a pathologic cavity lined by an epitheliated sac and containing fluid or semisolid material.

I. *Developmental cysts* of the jaws are closed epithelium-lined sacs, within the jaws, containing fluid or semifluid and derived from ectodermal remnants.

A. From odontogenetic tissue:

- 1. *Periodontal cysts* are those closed, epithelium-lined sacs, formed in the periodontal membrane and adjacent structures, usually at the periapex of a tooth but sometimes along the lateral root surface, which derive their epithelium from remnants of the Sheath of Hertwig or the dental lamina.

Synonyms: Dentoalveolar, dentoperiosteal, radicular, root end, dental root, paradontal, etc., cyst.

These cysts may be subdivided into the *dental root apex* or *radicular type* which occurs at the periapex and which is usually

\*A classification similar to this was submitted for comment and suggestions to a number of surgeons, radiographers, and pathologists. Constructive comment and criticism received has been most valuable in arriving at the classification presented here. Therefore, this classification is not the work of the author alone, but, in its present form, represents joint effort with these collaborators: V. P. Blair (Washington University), Paul Boyle (University of Pennsylvania), Lester Cahn (Columbia University), Henry Goldman (Army Medical Museum), E. H. Hatton (Northwestern University), Thomas J. Hill (Western-Reserve University), R. H. Ivy (University of Pennsylvania), D. A. Kerr (University of Michigan), Paul C. Kitchin (Ohio State University), William E. Koch, Jr. (Washington University), Sterling Mead (Washington, D. C.), Balint Orban (Loyola University, Chicago), B. G. Sarnat (St. Louis), H. Sicher (Loyola University, Chicago), C. O. Simpson (St. Louis), Harold A. Solomon (N. Y. State Institute for Study of Malignant Disease), Harry D. Spangenberg (Ohio State University), E. C. Stafne (Mayo Clinic), Kurt H. Thoma (Harvard University), Carl W. Waldron (University of Minnesota).

preceded by chronic proliferative inflammation (dental granuloma) and contains or contacts a tooth root; the *lateral type* which occurs laterally along the root surface; and the residual type which may be left after the extraction of a tooth with either a radicular or lateral type cyst or which may arise from epithelium in the region of residual infection. The cysts arising laterally to the tooth roots anterior to the first molar and containing cementum and dentine structures may be variants of periodontal cysts at the retained roots of deciduous teeth.

2. *Dentigerous cysts* are closed epithelium-lined sacs formed about the crowns of unerupted teeth or dental anomalies. They always contain tooth crowns or tumorous anomalies (odontomes). They have been described as originating through breakdown of the stellate reticulum of the enamel organs of developing teeth, but most of them probably are results of degenerative changes in reduced enamel epithelium. So-called eruption cysts, which occur especially on the distal of third molars, are dentigerous cysts that have developed at a comparatively late stage of odontogenesis.

Synonym: Follicular cyst (see *Discussion*).

3. *Primordial\** cysts are those closed epithelium-lined sacs formed through degeneration of the stellate reticulum in enamel organs before any calcified structures have been laid down. They contain no calcified structures.

Synonyms: Dentigerous cyst, follicular cyst (see *Discussion*).

#### B. From nondental tissues:

1. *Median cysts* are closed epithelium-lined sacs formed in the median fissure of the palate from embryonal remnants. They are situated between the two halves of the palate.
2. *Globulomaxillary cysts* are epithelium-lined sacs formed between the globular (median palatine) process and the maxillary process from embryonal remnants.
3. *Incisive canal cysts* (nasopalatine cysts) are closed epithelium-lined sacs formed within the incisive canal from embryonal remnants. A variant of this cyst occurs in the incisive foramen without involving the rest of the canal and may be called a cyst of the palatine papilla.<sup>14</sup>

#### II. Cystic neoplasm of dental origin:

*Ameloblastomas* are embryonal neoplasm derived from cells with a potentiality for enamel formation (oral epithelium, dental laminae, or embryonal amelogenic remnants). They are first solid, mimicking the developing amelogenic tissues, but later become cystic at the expense of their stellate cells at the centers of the tumor follicles.

Synonyms: Adamantinoma, adamantinoblastoma, epithelial odontoma, multilocular cyst, etc.

#### DISCUSSION

The only four types of cysts formed from the dental structures are periodontal cysts, dentigerous cysts, primordial cysts, and ameloblastomas (Fig. 1).

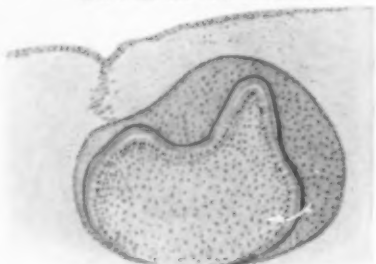
\*"Original or primitive; of the simplest and most undeveloped character."

## ODONTOGENESIS

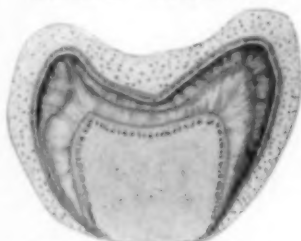
PROLIFERATION



DIFFERENTIATION



TISSUE FORMATION



TISSUE FORMATION



ERUPTED TOOTH



## CYSTS AND NEOPLASM

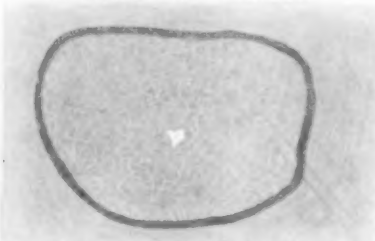
SOLID AMELOBLASTOMA



CYSTIC AMELOBLASTOMA



PRIMORDIAL CYST



DENTIGEROUS CYST



PERIODONTAL CYST



## ORIGIN OF LESIONS

THE AMELOBLASTOMA IS AN EPITHELIAL NEOPLASM WHICH RESEMBLES DENTAL LAMINAE AND ENAMEL ORGANS UNTIL THE PERIOD OF AMELOGENESIS. IT MAY BE DERIVED FROM CELLS OF THE ORAL EPITHELIUM WITH A TENDENCY TO ODONTOGENESIS, FROM REMNANTS OF THE SHEATH OF HERTWIG OR THE DENTAL LAMINA (EPITHELIAL RESTS) OR FROM ABERRANT TOOTH BUDS. IT BEGINS AS A SOLID TUMOR APING THE DENTAL ANLAGE AND ENAMEL ORGAN BUT NEVER FORMS ENAMEL. IT DEGENERATES AT THE EXPENSE OF THE STELLATE RETICULUM TO BECOME A MULTICYSTIC TUMOR.

THE PRIMORDIAL CYST IS A CYST OF THE JAW DERIVED FROM THE ENAMEL ORGAN IN ITS EARLY STAGES. BEFORE TISSUE FORMATION BEGINS, THE STELLATE RETICULUM BREAKS DOWN AND FLUID COLLECTS BETWEEN THE INNER AND OUTER ENAMEL EPITHELIUM. THE CYST IS FORMED BY INTERNAL PRESSURE.

THE DENTIGEROUS CYST IS A CYST OF THE JAW CONTAINING THE CROWN OF A TOOTH. IT IS USUALLY DESCRIBED AS FORMED BY A BREAKDOWN OF THE STELLATE RETICULUM DURING AMELOGENESIS. THIS WOULD PRODUCE HYPOPLASTIC ENAMEL. IT APPEARS TO BE FORMED WITHIN THE REDUCED ENAMEL EPITHELIUM.

THE PERIODONTAL CYST IS A CYST FORMED IN THE PERIODONTAL MEMBRANE USUALLY AT THE ROOT END OF A PULPLESS INFECTED TOOTH. THE EPITHELIAL LINING IS DERIVED FROM THE EPITHELIAL RESTS (USUALLY REMNANTS OF THE SHEATH OF HERTWIG). THEY ARE COMMONLY THE SEQUELS OF DENTAL GRANULOMATA, IN WHICH EITHER RESTING OR PROLIFERATING EPITHELIUM IS A CONSTANT FINDING.





They are all of developmental origin although trauma, infection, or other stimulation may play a role in stimulating their epithelial proliferation. In the instance of the periodontal cyst, which is commonly preceded by chronic inflammatory reaction, the role of irritation acting as a stimulating factor is especially prominent. For this reason periodontal cysts have often been classified as "inflammatory cysts." While inflammation appears to afford the stimulus, the epithelium which proliferates is "senile" amelogenic remnant.

The dentigerous and follicular cysts have been considered as cysts of one type by many authorities. Although they both arise from amelogenic epithelium, they arise at different periods in the developmental career of the enamel-forming tissues, the follicular cyst during histodifferentiation and the dentigerous cyst after apposition of the enamel. They are readily differentiated radiographically and histologically by the presence or absence of calcified structures within their cavities. If we consider the soft connective tissue surrounding an unerupted tooth as the tooth follicle, the term follicular cyst is seen to be misleading. Not the follicle, but its epithelial content is involved. For this reason the term follicular cyst has been discarded. The primordial cysts may occur in single or multiple form and the enamel organ of a single tooth of the regular series may be the only contributing tissue, or numerous aberrant dental anlage may become cystic. While a few dentigerous cysts may begin their formation by stellate reticulum degeneration during enamel apposition, a far greater number begin to form after the enamel apposition is completed and the reduced enamel epithelium is present. Those formed during enamel apposition should contain hypoplastic enamel, for the same pathologic process that would initiate degenerative changes in the stellate reticulum would also result in pathologic enamel apposition. The lining of both primordial and dentigerous cysts is stratified squamous epithelium.

The multiple type of primordial cyst has been described as occurring sporadically and as a familial disease.<sup>15</sup> It might be suggested that these cysts arise from overgrowth of odontogenetic epithelium, differing from that of ameloblastomatous growth by degree. In one instance cysts develop, in the other, tumor follicles.

The ameloblastoma rapidly changes from its solid stage with differentiation to dental lamina and enamel organlike structures, to its multicystic stage. Its solid to cystic transition has been noted in series of cases<sup>7, 8</sup> and in a single individual.<sup>12</sup> Zegarelli<sup>18</sup> has stated that adamantinoblastomas (ameloblastomas) originate from the outer enamel epithelium. This conclusion, derived from study of ninety-eight jaw tumors in a cancer strain of mouse, does not appear justified. The tumors that he illustrates do not show the typical picture of ameloblastomas (tall columnar cells bordering follicles with stellate cells centrally) but appear more like dentigerous cysts, epitheliomas, and adenocarcinomas. Moreover, this origin would not explain the high degree of differentiation toward ameloblasts observed in many solid ameloblastomas, is not compatible with the origin from gingival tissue sometimes strongly suggested in human ameloblastomas, and is incompatible with the usual explanation of hypophyseal ameloblastomas. While the enamel organlike tissues of the ameloblastoma do not become functional, they do have the characteristics of dental lamellae and enamel organs up to the point of function. The epithelium lining the cysts of ameloblastomas may

vary from columnar, through cuboidal, to squamous type, but the tissue arrangement remains characteristic. A question may be raised as to placing ameloblastomas in a separate group from other cysts derived from odontogenic tissues. While the ameloblastoma is from the same general primordium as the periodontal, dentigerous, and primordial cysts, it develops as a neoplasm which becomes cystic, not primarily as a cyst, and for this reason is considered separately.

The epithelium lining of any of these cysts may, under certain stimulation, become anaplastic and result in squamous cell carcinoma. The ameloblastoma becomes malignant in about 4.5 per cent of the cases<sup>9</sup> and the others probably in lesser percentages. Repeated surgical intervention appears to increase the possibility of change to malignant neoplasm.

The developmental cysts of nondental origin may be misdiagnosed as dental cysts when they are adjacent to dental roots.<sup>11</sup> The epithelial lining in median palatal cysts is usually of squamous type but they may be lined, in part, by ciliated columnar epithelium contributed from the nasal side. There are occasionally cysts appearing between the roots of the maxillary first incisors, or more rarely between the roots of the mandibular first incisors. Since the portions of the maxillary and mandibular bones which meet in the midline arise from centers of ossification deep within the mesenchyme, there is no opportunity for epithelial inclusion between these bone segments, and true fissural cysts cannot develop in these regions. The cysts which occasionally appear at these sites are probably primordial cysts. Those in the maxilla may develop from analage of supernumerary teeth (mesiodens of Bolk).

The extravasation "cysts" (hemorrhagic, traumatic) represent an unusual group in that they are not lined by epithelial tissue but have fibrous membranes lining their cavities. They may follow blows on the jaws which produce hemorrhage without fracture and are therefore more common in young individuals with more plastic osseous structures, and most commonly occur near the angle of the mandible. A possible variant of this cyst is the traumatic osteoclasia described by Simpson<sup>13</sup> at the apices of vital mandibular incisors subjected to excessive occlusal stress. This lesion may resemble cementomas, but the author is convinced that both cementomas and traumatic osteoclasia exist as entities.

Any of a large group of neoplasms may simulate cysts of unilocular or multilocular form, radiographically. Among these are the central giant cell tumor, epithelioma arising from the gingival epithelium or dental embryonal remnants, adenocarcinoma, metastatic carcinoma, Ewing's tumor, fibro-osteoma or localized osteitis fibrosa, sarcoma, and mixed tumors. Metabolic dysfunctions, including osteitis deformans (fibrous stage), multiple myeloma, osteitis fibrosa cystica (von Recklinghausen's disease), and xanthomatoses, may also destroy bone and produce the appearance of cysts. Osteomyelitis, actinomycosis, and syphilis may produce inflammatory lesions that appear cystic. All of these non-cystic lesions are readily differentiated from the true cysts by histologic examination.

Ranula, dermoid cyst, branchial cleft cyst, nasoalveolar cyst, thyroglossal cyst, and cysts of the salivary and mucous glands are not included in this classification because they rarely or never involve the tooth-bearing bones and because they seldom present problems of differential diagnosis from the lesions under discussion.

## SUMMARY

The cysts of the jawbones are divided into developmental cysts from odontogenic tissue (periodontal, dentigerous, and primordial cysts), developmental cysts of nondental origin (median, globulomaxillary, and incisive canal cysts) and ameloblastomas. These cysts are defined and a few subtypes cited. The extravasations, neoplasms, metabolic dysfunctions, and inflammations that may simulate these cysts are mentioned.

Differential diagnosis will be discussed in a subsequent publication.

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## A THIMBLE INSTRUMENT HOLDER

JEROME J. PAVLIGER, D.D.S., OAKLAND, CALIF.

IN ALL lines of production today, methods to increase speed and efficiency are welcomed. In dentistry, too, because the operator is working under greater pressure, we are seeking ways to speed up our techniques and yet reduce the strain.

A thimble instrument holder which I devised seems to help in this way. By slipping an instrument, such as a dental mirror, through a holder attached to a thimble, this instrument can be manipulated by one finger, leaving the other fingers free for other purposes. This may make it possible that the dental surgeon may hold both a mirror and a retractor in one hand, while using an exolever, chisel, or drill with the other.

*Description of Instrument.*—A metal tube is soldered to a metal thimble which has previously been chosen for finger size. Various instruments may be slipped through this tube, such as indicated in Fig. 1.



*Technique.*—The thimble is worn on one finger, allowing the free use of the other fingers of that hand. The mirror or working part of the instrument should be kept close to the end of the tube. When using the holder for a mouth mirror, the handle of the mirror may be left off for ease of movement.

*Function With a Mirror.*—Many situations in the mouth do not permit the direct reflection of the rays of light to the eyes without assuming positions of the body and of the head of the operator which are awkward to free movement of the hand, as well as necessitating inconvenient and tiresome positions of the head of the patient.

This method of working is at first difficult to the novice, but by continued effort it becomes as easy to make correct application of movements by this method as by direct rays of light.

Further continued practice in this way renders the movements so completely under reflex control that the operator passes from a direct movement to a reverse one, and the contrary, without an apparent effort of the brain. This is equally true in all the various movements, even of those where considerable force is required to be employed.



Fig. 1.

*Delicacy of touch.*—That one should acquire a deft touch in the holding and manipulating of instruments was advocated by G. V. Black in his texts on operative dentistry. "Facility and delicacy of touch and movement are acquired by careful training and observation," he states. "These are easily lost by careless habits. The man who makes use of delicate movements acquires a much finer appreciation of size and form than the man who does not acquire this finger dexterity." Delicacy of touch is especially important in retracting tissue during oral surgical operations. If the operator holds the retractor himself, he does less damage than an assistant who may not be able to see what he is doing or may get tired and then put too much strain on the instrument, thus causing a great deal of postoperative swelling and pain.

The use of the mouth mirror also decreases fatigue of the operator. If adequately trained in the use of the mouth mirror, he seldom has reason to stand long in any position while operating. By standing erect, beside and slightly behind the patient's right shoulder, very little twisting or bending of the back is required. This also provides for a more effective arm and hand control so deftness and conservation of action is facilitated.

*Conclusion.*—An instrument holder requiring the use of one finger to manipulate and freeing the other fingers of that hand for other uses fits in with today's need for increased speed in technique and prevention of tissue injury. With many facing lack of help from assistants, a device of this kind is well worth the effort to construct and master its use.



## Case Reports

CASE NO. 93

### CORRECTION OF A CLEFT PALATE USING A STRESS-BREAKER TYPE\* PARTIAL APPLIANCE WITH OBTURATOR ATTACHED

O. E. BEDER, B.S., D.D.S., W. J. MILLER, A.B., D.D.S., G. P. SMITH, D.D.S.,  
AND D. E. ZISKIN, D.D.S., NEW YORK, N. Y.

**M**R. W. S., white, aged 36 years, was referred to our institution by his dentist for the correction of his cleft palate.

The history discloses that attempts to correct the defect by surgical means had failed. The patient's medical history was negative.

The patient had marked nasality of speech and complained that he had difficulty in eating.

Oral examination revealed the following: The patient had a Class V Cleft (Fig. 1), i.e., there is a complete separation of the tissues and bone beginning in the uvula, which is divided, and ending in the alveolar ridge mesial to the left cuspid (at junction of maxilla and premaxilla). The unilateral harelip which accompanied the cleft palate was successfully repaired some years previous to presentation (Fig. 2). The cervicals of the remaining teeth were covered with food and debris, indicating very poor hygiene.

The missing teeth were the upper left third, second, and first molars, second and first premolars, central and lateral incisors; upper right first and second premolars and first molar; lower left third and first molars, second premolar, central and lateral incisors, lower right central and lateral incisors, first premolar, first, second, and third molars. The occlusion was of Class III type (Angle's Classification). The lower jaw protruded and deviated slightly to the left.

There was a lower anterior fixed bridge from cuspid to cuspid replacing the lateral and central incisors. The bridge was composed of two cast full crowns and four pontics. The patient had worn an obturator in the past.

#### INDIVIDUAL TEETH

The upper left cuspid had a full crown with a porcelain facing which was defective. The upper left first premolar root showed evidence of periapical infection in the x-ray. The second premolar had a full crown; it was nonvital with evidence of periapical infection. The upper left first molar, consisting of partially embedded roots, and the second molar showed evidence of periapical infection in the x-ray.

The upper right cuspid had large mesial and distal cavities and was extruded. The upper right second molar had a large occlusal amalgam filling which was passable. The third molar had an occlusal cavity.

The lower left second molar had an occlusal amalgam which was defective and was also carious. The lower left first premolar had mesial and occlusal cavities. The lower cuspids had defective full cast gold crowns serving as abutments for the fixed bridge.

The lower right first premolar consisted of partially embedded roots with evidence of periapical infection. The lower right second premolar had occlusal, mesial, and buccal cavities. The second molar consisted of partially embedded roots with evidence of periapical infection.

The patient was treated by the following procedures: (1) surgery, (2) crown and bridge restoration, (3) prosthesis.

\*A stress-breaker type denture was demonstrated to us by Dr. George Stein, Columbia University School of Dental and Oral Surgery.

*Surgery.*—The following teeth or roots were removed: upper left first premolar root, upper left second premolar, upper left first molar roots, upper left second molar, lower right first premolar roots, and lower right second molar roots.

*Crown and Bridge Restoration.*—The following teeth were excavated, sterilized, and filled with red copper cement where necessary, and were covered with full crowns having lug

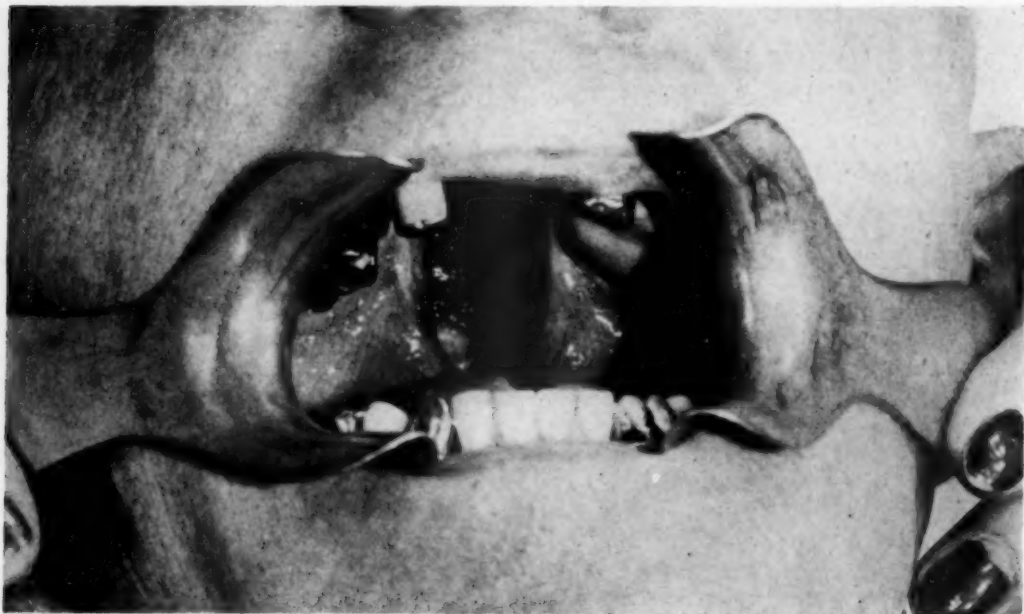


Fig. 1.



Fig. 2.

seats for rests; upper left cuspid, upper right cuspid, upper right second and third molars; lower left second molar, lower left first premolar, lower left cuspid, lower right cuspid, and lower right second premolar.

*Prosthesis.*—A lower partial denture was constructed, consisting of a cast gold harness with circumferential clasps on all teeth except the lower cuspids. The missing teeth in

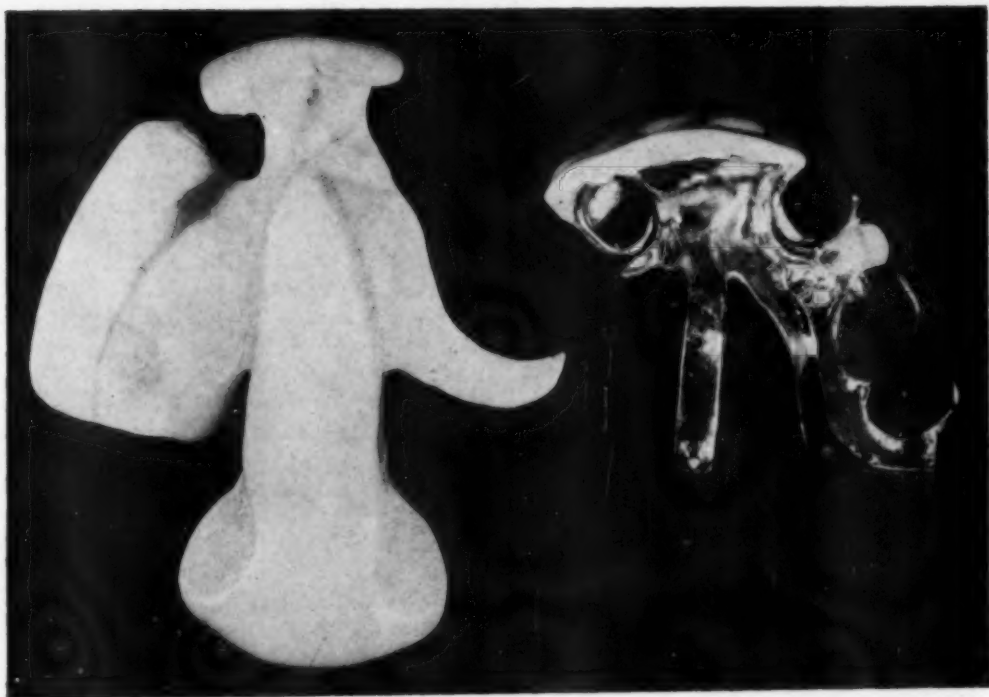


Fig. 3.

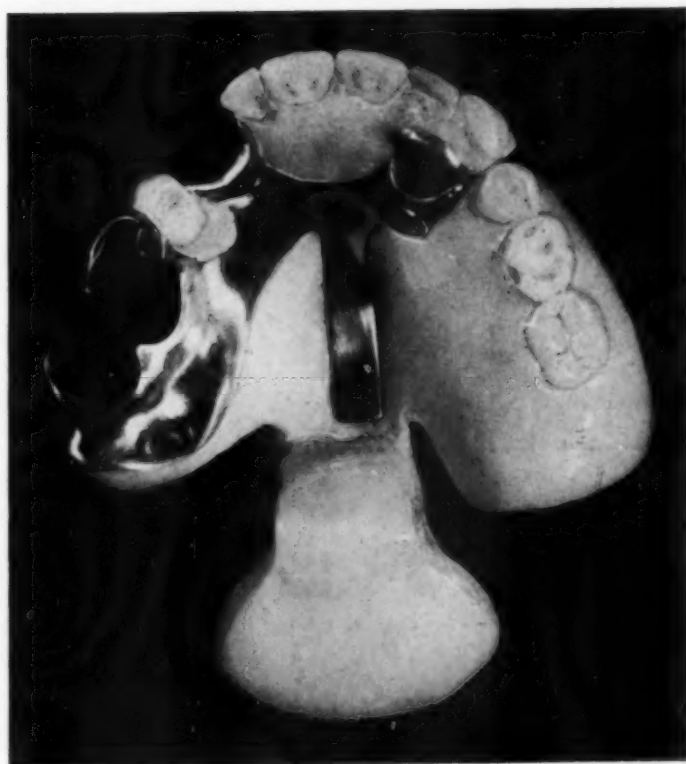


Fig. 4.

the edentulous areas were replaced in acrylic saddles attached to the harness.

In the upper jaw, the main goal was the correction of the cleft palate. However, this was complicated by the poor distribution of the upper teeth, which made a stress-breaker partial denture necessary. For this reason the upper appliance was made in two portions (Fig. 3); one portion was tissue-borne and the other tooth-borne. The tooth-borne entity consisted of a cast gold harness having circumferential clasps on the cuspids and one on the upper right second molar and an arm engaging the distobuccal undercut of the upper right third molar. There were rests on the labial and mesial surfaces of the upper left cuspid, lingual of the upper right cuspid, and mesio-occlusal surface of the upper right second molar. The harness of the tooth-borne entity carried a bar which extended through the center of the cleft at the approximate level of the palate. This bar acted as an additional support for the tissue-borne member along with the acrylic overlap area on the lingual of the tooth-borne portion. The tooth-borne member replaced the upper left cuspid labial surface, the upper incisors, and the upper right first premolar. The tissue-borne member replaced the upper left posteriors, closed off the defect of the hard palate, and carried the artificial velum. It was held in place by an overlapping flange of the metal together with the lingual of the upper right side and the central bar which fitted into a groove in the acrylic (Fig. 4). Such a device provided the stress-breaking necessary to allow movement of the tissue-borne member toward the ridge but prevented movement away from the ridge. The bar of the tooth-borne member prevented lateral movement of the tissue-borne section. The manner of fixation of the tissue-borne section provided sufficient rigidity for the artificial velum, which was of the fixed type, modified after Dr. Fitzgibbon's speech appliance.

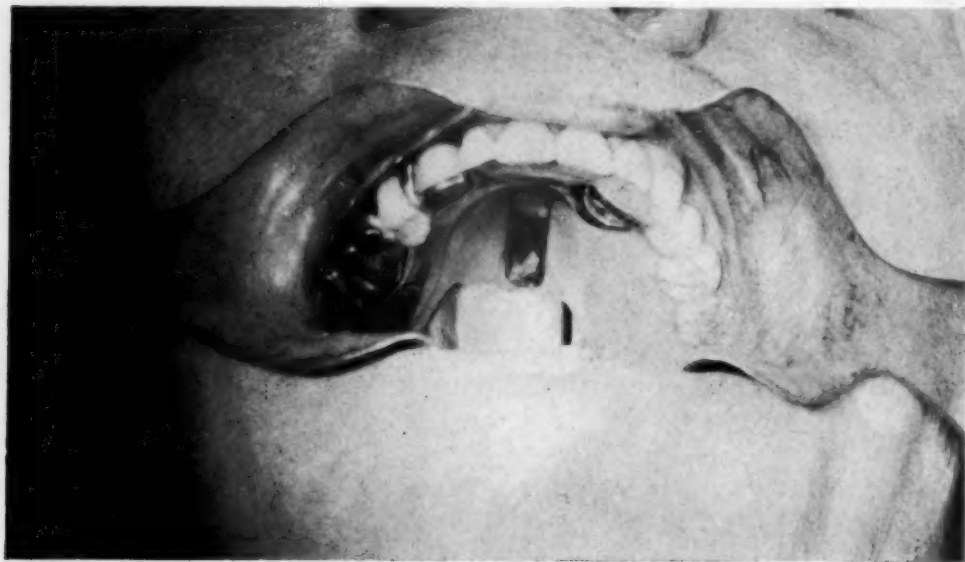


Fig. 5.

The velum was constructed as follows: An apron which followed the contour of the soft palate at rest was constructed so that its dimensions approximated the soft palate up to the uvula. A loop of 16 gauge round wire was next added to the distal end, which pointed toward Passavant's pad. Next, some baseplate wax was fashioned around the loop, short of the dimensions of the muscles of the soft palate in function. Lastly, some carding wax was added to the other wax, softened, and the appliance inserted. This was then trimmed by the muscles in function by having the patient swallow, move the head about, etc. This was adjusted until a perfect seal was obtained. The velum was finally converted into acrylic by the usual methods.

The appliance (Fig. 5) served to close the cleft in the palate as well as to restore normal function and esthetics. The patient has worn it for over a year with considerable success. The most notable remedial effect was in his speech which returned almost to normal. He can eat with greater comfort now.





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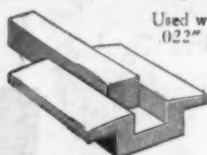
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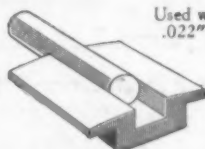
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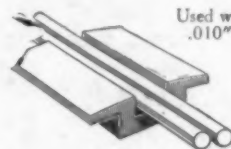
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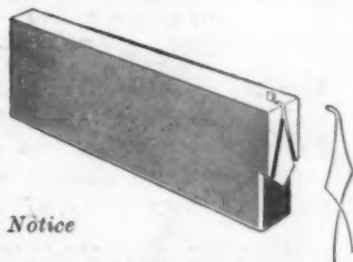
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